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Urban Soil Lead Abatement Demonstration Project

Volume I:
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National Center for Environmental Assessment
Office of Research and Development
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Research Triangle Park, NC 27711

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LIST OF ABBREVIATIONS, ACRONYMS, AND TERMS

AAS	Atomic absorption spectroscopy
ANCOVA	Analysis of covariance
BAL P1, BAL P2	Baltimore Study Group with paint intervention
BAL SP	Baltimore Study Group with soil and paint intervention
BOS P-S	Boston Study Group with paint intervention, followed by soil abatement in second year
BOS PI-S	Boston Study Group with paint and interior dust intervention, followed by soil abatement in second year
BOS SPI	Boston Study Group with soil, paint, and interior dust intervention
CDC	Centers for Disease Control and Prevention
CIN I-SE	Cincinnati Study Group with interior dust intervention, followed by soil and exterior dust intervention (second year)
CIN NT	Cincinnati Study Group with no treatment
CIN SEI	Cincinnati Study Group with soil, exterior dust, and interior dust intervention
dL	Deciliter; used here as a measure of blood lead in micrograms per deciliter
Double blind	Analytical audit sample where analyst knows neither that the sample is an audit sample nor the concentration
Dust loading	Mass of dust per unit area
ECAO/RTP	Environmental Criteria and Assessment Office/Research Triangle Park (now National Center for Environmental Assessment/Research Triangle Park)
EPA	U.S. Environmental Protection Agency
GLIM	Numerical Algorithms Group software package for a general linear model

GLM	SAS procedure for general linear models approximately equivalent to Systat MGLH
Hand dust	Sample taken by wiping the child's hand thoroughly; a measure estimating the ingestion of lead
HEPA	High-efficiency particle accumulator
ICP	Inductively coupled plasma emission spectroscopy
Lead concentration	Mass of lead per mass of medium (soil, dust, water)
Lead loading	Mass of lead per unit area
MGLH	Systat procedure for general linear models approximately equivalent to SAS GLM
NHANES II	National Health Assessment and Nutrition Examination Survey II
ORD	Office of Research and Development
OSWER	Office of Solid Waste and Emergency Response
P-value	Statistical term for the likelihood that an observed effect differs from zero
Pb	Lead
Project	In this report, "project" refers collectively to the three individual studies that compose the Urban Soil Abatement Demonstration Project.
P-XRF	Field or Portable XRF used in this study for paint measurements
QA/QC	Quality assurance/quality control
Repeated measures analysis	Statistical procedure for analyzing normally distributed responses collected longitudinally
Round	Period of sampling and data collection during study
SARA	Superfund Amendments and Reauthorization Act

1. EXECUTIVE SUMMARY

1.1 BACKGROUND AND OVERVIEW

During the past 25 years, concern for lead toxicity in children has steadily increased with mounting evidence for the subtle but serious metabolic and developmental effects of lead exposure levels previously thought to be safe. Childhood lead poisoning was formerly considered a severe medical problem usually traced to swallowed chips of peeling lead-based paint. Scientific evidence has systematically revealed deleterious effects of environmental lead at lower levels of exposure. Federal agencies such as the U.S. Environmental Protection Agency (EPA) and the Centers for Disease Control and Prevention (CDC) have repeatedly lowered the level of concern for children's lead burden that recommends environmental or clinical intervention—from a blood lead level of 30 μg/dL established in 1978 by CDC to 25 μg/dL in 1985 (just prior to the start of this project), then to the present level of 10 μg/dL (defined in October 1991 by CDC as a blood lead level that should trigger community-wide prevention activities if found in many children).

The Urban Soil Lead Abatement Demonstration Project (USLADP), known also as the "Three City Lead Study", was authorized in 1986 under Section 111(b)(6) of the Superfund Amendments and Reauthorization Act (SARA), which mandated that EPA conduct soil lead abatement projects in up to three U.S. cities (SMSA's). The purpose of the project was to determine whether abatement of lead in soil could reduce the lead in the blood of inner city children. It did not attempt to compare the relative effectiveness of alternative soil abatement methods.

The project began in December 1986 with the appointment of a U.S. EPA steering committee to develop recommendations for implementing the SARA lead-in-soil abatement demonstration project. A panel of experts was formed in early 1987 to assist U.S. EPA in defining a set of criteria for selection of sites and the minimum requirements for a study at each site. The panel also met in mid 1987 to discuss technical issues and study designs and to evaluate technical criteria for selection of urban areas as potential soil-lead abatement demonstration project sites, ultimately leading by the end of 1987 to the selection of Boston, Baltimore, and Cincinnati as the participating cities.

The individual studies in each city were designed around the concept of participating families within a definable neighborhood. These families and their living units were part of a study group, either a treatment group or a control group. Each study group was sampled during preabatement and postabatement phases of the studies carried out in each city. Prior to and after abatement, blood lead levels were ascertained and the environment of the child was extensively evaluated through measurements of lead in soil, dust, drinking water, and paint, and through questionnaires about activity patterns, eating habits, family activities, and socioeconomic status (SES). Because of the complex nature of this exposure assessment, intermediate exposure indices, such as street dust, house dust, and hand dust were measured in some study groups. The objective of the preabatement phase was to determine the baseline exposure history and status (stability of the blood lead and environmental measures) prior to abatement. During the postabatement phase, samples were taken to confirm the effectiveness of abatement actions in reducing lead in the abated media, to measure the duration of the effect of soil abatement, and to detect possible recontamination. Blood lead measurements were also obtained postabatement to ascertain abatement impacts at various postabatement intervals.

Research teams in each city included state and/or local health department personnel, academic researchers from local universities, and/or various other institutions (including in Boston participation by U.S. EPA Region I Laboratory personnel). Protocols for the environmental and blood lead measurements were developed by a Scientific Coordinating Committee composed of representatives from each city's research team, three pertinent EPA Regional Offices (I, III, V), EPA/Office of Solid Waste and Emergency Response, EPA/Office of Research and Development (ORD), and the CDC. Lead responsibility for coordinating technical oversight for the project fell to EPA/ORD. This was accomplished mainly via a series of workshops (2 to 3 per year) organized by ORD's Environmental Criteria and Assessment Office in Research Triangle Park, NC (ECAO/RTP)[1], at which efforts were made to standardize measurement methods across the three individual city studies, compare approaches to statistical analyses used by each research team, and,

[1]ECAO/RTP, formerly within ORD's Office of Health and Environmental Assessment (OHEA), is now a unit within ORD's National Center for Environmental Assessment (NCEA).

ultimately, to obtain external peer review of the results of the studies contained in the individual city reports[2].

This report, then, is an integrated assessment of data from the above-noted coordinated longitudinal studies of children in urban neighborhoods of three cities (Boston, Baltimore, Cincinnati), where intervention into soil lead exposure pathways was expected to reduce the children's blood lead. Many cross-sectional studies of childhood lead exposure have previously shown that differences in soil lead exposure are associated with differences in blood lead concentrations, but they did not evaluate the effectiveness of intervention steps in terms of demonstrating that reductions in external exposure to lead from soil result in reductions in blood lead concentrations. Thus, a unique aspect of this project is that it measures response to intervention, not to contamination. Because of the physiology of lead mobilization in body tissues, there is a difference between the rate of change in a population with increasing lead exposure and in one with decreasing exposure. In other words, the decrease in blood lead concentrations in response to intervention was not expected to be at the same rate as an increase in blood lead concentrations in response to increasing exposure.

The relationship between soil lead and blood lead is an indirect relationship in the sense that children most commonly do not eat soil directly, but rather they mainly ingest small amounts of dust derived, in part, from soil. In the child's environment, dust is only one of several sources of lead that also include food, air, and drinking water. Likewise, the lead in blood reflects not only recent exposure from these sources but also the lead from accumulated body stores in bone and other tissues, which is released to blood by biokinetic processes that distribute and redistribute lead between blood and other body tissues.

1.1.1 Comparison of Study Hypotheses

The Scientific Coordinating Committee attempted to establish uniformity among the three studies for major aspects of the project. This required a study plan from each city that was discussed and reviewed at several early planning workshops. Although there were differences in form and content, each study plan contained

- a statement of the objectives of the study;

[2]Each of the three individual city reports prepared by the research teams from Boston, Baltimore, and Cincinnati, are appended to this U.S. EPA Integrated Report.

- a testable hypothesis that provided direction and focus to the study;
- protocols for collecting and analyzing the data;
- an array of treatment groups that addressed all features of the hypothesis;
- measures to be taken to ensure that all phases of the study would be conducted as planned; and
- procedures by which the results of the study would be processed, analyzed, and interpreted.

The objectives, protocols for sampling and analysis, quality assurance/quality control (QA/QC) plans, and data processing procedures were nearly identical for all three studies. Elements that differed among the three studies were the hypotheses and the varying array of treatment groups. The hypotheses differed only slightly, as seen from the following statements.

The overall central hypothesis of the USLADP is:

> *Reduction of lead in residential soil accessible to children will result in a decrease in their blood lead concentration.*

The formal statement of the Boston hypothesis is:

> *A significant reduction (equal to or greater than 1,000 μg/g) of lead in soil accessible to children will result in a mean decrease of at least 3 μg/dL in the blood lead levels of children living in areas with multiple possible sources of lead exposure and a high incidence of lead poisoning.*

The initial Baltimore hypothesis, stated in the null form, was:

> *A significant reduction of lead (≥ 1,000 μg/g) in residential soil accessible to children will not result in a significant decrease (3 to 6 μg/dL) in their blood lead levels.*

The Baltimore hypothesis, based on actual residential soil lead values averaging less than 1,000 μg/g, was later revised by U.S. EPA for statistical analyses purposes:

> *A one-time reduction of at least 500 ppm in the maximum lead concentration in yard soil, even when not accompanied by abatement of household dust or lead paint inside the child's apartment or residence unit, will not result in a reduction of blood lead in children living in housing in which exterior lead paint has been stabilized.*

The Cincinnati hypothesis was separated into two parts:

(1) *A reduction of lead in residential soil accessible to children will result in a decrease in their blood lead levels.*

(2) *Interior dust abatement, when carried out in conjunction with exterior dust and soil abatement, would result in a greater reduction in blood lead than would be obtained with interior dust abatement alone, or exterior dust and soil abatement alone.*

Secondary hypotheses in the Cincinnati study are:

(3) *A reduction of lead in residential soil accessible to children will result in a decrease in their hand lead levels.*

(4) *Interior dust abatement, when carried out in conjunction with exterior dust and soil abatement, would result in a greater reduction in hand lead than would be obtained with interior dust abatement alone, or exterior dust and soil abatement alone.*

The array of treatment groups differed considerably among the three studies (Table 1-1). In each study, the treatment groups had several features in common. The groups were taken from demographically similar neighborhoods. All groups had some prior evidence of elevated lead exposure, usually a greater than average number of public health reports of lead poisoning. Three phases were employed in each study: a preabatement baseline phase for 3 to 18 mo; an abatement or intervention (except for controls) phase; and a postabatement follow-up for 10 to 23 mo.

1.1.2 Study Design and Conduct

Table 1-1 describes the study groups and the forms of intervention employed in each of the three cities. The Cincinnati study design used intervention on the neighborhood scale, where the soil in parks, play areas and other common grounds were abated, and paved surfaces in the neighborhood were cleaned of exterior dust. In Boston and Baltimore, only soil on individual properties was abated. Table 1-2 shows the number of subjects participating in different phases of the three studies in relation to the respective participant groups for each city. The general characteristics are that soil lead concentrations are typically high in Boston, where it is also common to find lead in both exterior and interior

TABLE 1-1. DESCRIPTION OF STUDY GROUPS AND TYPES OF INTERVENTION

Treatment Group Name[a]	Cross-Reference to Individual Study Report	Description of Treatment
		BOSTON
BOS SPI	Study Group	Soil and interior dust abatement, and interior paint stabilization at beginning of first year, no further treatment.
BOS PI-S	Control Group A	Interior dust abatement and interior paint stabilization at beginning of first year. Soil abatement at beginning of second year.
BOS P-S	Control Group B	Interior paint stabilization at beginning of first year. Soil abatement at beginning of second year.
		BALTIMORE
BAL SP	Study Area	Soil abatement and exterior paint stabilization at beginning of first year, no further treatment.
BAL P1[b]	Control Area	Exterior paint stabilization at beginning of first year, no further treatment.
BAL P2[b]	Study Area Not Abated	Exterior paint stabilization at beginning of first year, no further treatment because soil not above cut-off level.
		CINCINNATI
CIN SEI (P)	Area A	Soil, exterior dust, and interior dust abatement at beginning of first year, no further treatment. Includes only the Pendleton neighborhood.
CIN I-SE (B,D,F)[c]	Area B	Interior dust abatement at beginning of first year, soil and exterior dust abatement at beginning of second year, no further treatment. Includes the Back St., Dandridge and Findlay neighborhoods.
CIN NT (G,M)[c]	Area C	No treatment, soil and interior dust abatement following last sampling round. Includes the Glencoe and Mohawk neighborhoods.

[a]The treatment group designation indicates the location of the study (BOS = Boston, BAL = Baltimore, CIN = Cincinnati), the type of treatment (S = soil abatement, E = exterior dust abatement, I = interior dust abatement, P = loose paint stabilization, NT = no treatment).

[b]Treated as one group in the Baltimore report, analyzed separately in this report.

[c]Treated as one group for many of the analyses in the Cincinnati report, analyzed as individual neighborhoods in this report.

TABLE 1-2. NUMBER OF PROJECT PARTICIPANTS BY ROUND[a]

Study						
BOSTON	Round 1	Round 2	Round 3	Round 4		
Middate	10/17/89	4/9/90	9/12/90	7/20/91		
Children[b]	150	146	147	92		
Famlies[c]	125	121	122	77		
Properties[d]	100	96	97	67		
BALTIMORE	Round 1	Round 2	Round 3	Round 4	Round 5	Round 6
Middate	10/25/88	4/1/89	2/17/90	1/27/91	6/7/91	9/3/91
Children[b]	168	165	198	190	186	182
Families[c]	119	116	131	126	122	122
Properties[d]	106	104	115	112	105	110
CINCINNATI	Round 1	Round 3	Round 4	Round 6	Round 7	
Middate	7/6/89	11/14/89	7/1/90	11/17/90	6/16/91	
Children[b]	201	185	219	198	169	
Families[c]	129	123	122	168	142	
Properties[d]	215	245	245	243	245	

[a]Number shown is based on samples taken and does not include individuals enrolled but not sampled. Intervention is shown by the vertical dashed lines.
[b]Based on number of children sampled for blood. Some children may not have been included in the statistical analyses.
[c]Based on number of households sampled for dust.
[d]Based on number of properties (Boston, Baltimore) or soil parcels (Cincinnati) sampled.

paint, as well as in drinking water. In the Boston areas studied, housing is typically single and multi-family units with relatively large lot sizes. In the Baltimore neighborhoods, the houses were mixed single and multifamily, and the lots were smaller than Boston lots, with typical yards less than 100 m^2. Nearly every house had lead-based paint. Residential units in Cincinnati were mostly multifamily with little or no soil on the residential parcel of land.

1.1.3 Intervention Procedures

Figure 1-1 illustrates the generalized concept of human exposure to lead, showing the pathways of lead from the several sources in the human environment to four compartments immediately proximal to the individual. In the past decade, dramatic reductions in exposure to lead in air and food have occurred as a result of regulatory and voluntary programs to reduce lead in gasoline and canned food. Figure 1-2 expands the critical dust pathway to show the complexity of the many routes of dust exposure for the typical child. The strategies for intervention used in this project were designed to interrupt the movement of lead along one or more of these dust pathways.

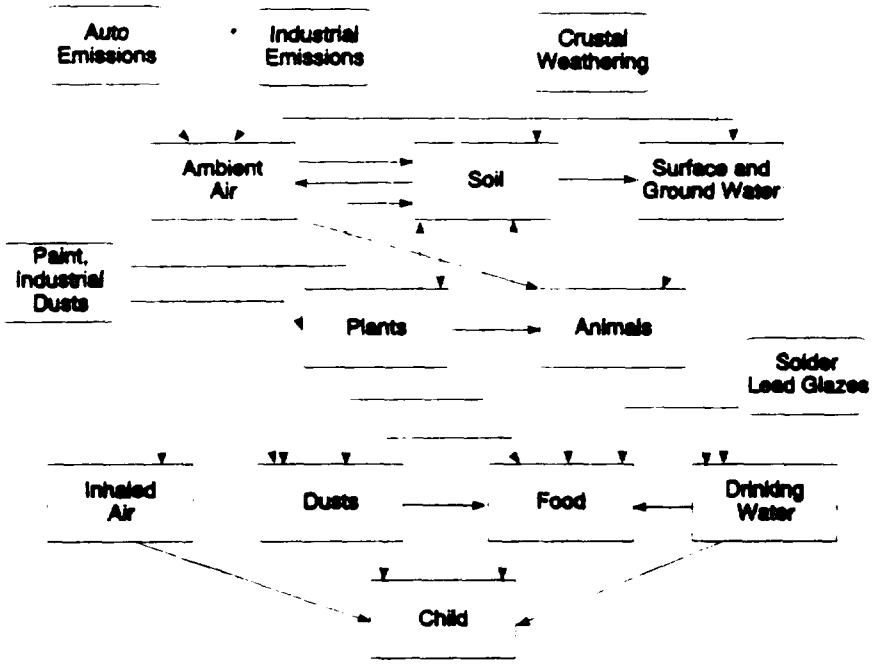

Figure 1-1. Generalized concept of the sources and pathways of lead exposure in humans.

There were three forms of intervention in this project: (1) soil abatement, (2) dust removal, and (3) paint stabilization. Soil abatement was by excavation and removal, followed by replacement with clean soil (<50 $\mu g/g$). Dust intervention was by vacuuming, wet mopping, and, in some cases, replacement of rugs and upholstered furniture. Cincinnati

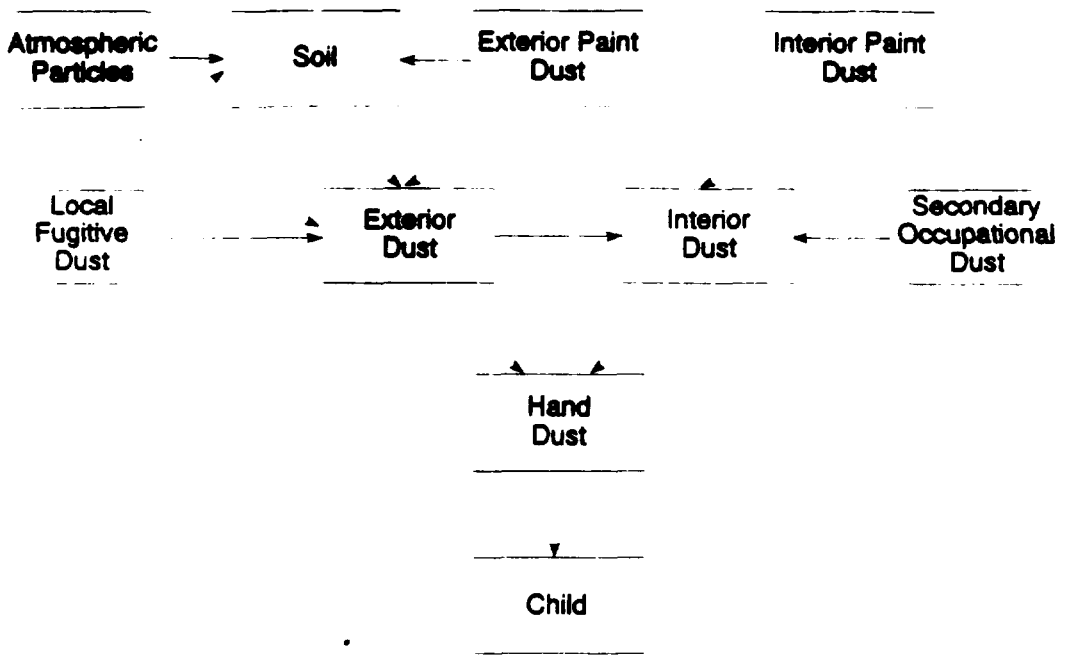

Figure 1-2. Typical pathways of childhood exposure to lead in dust.

and Boston performed interior dust abatement, and Cincinnati also removed neighborhood exterior dust with mechanical sweepers and hand tools. Dust intervention was not expected to be permanent, because dust continually moves through the human environment. Instead, the removal of dust with elevated lead concentrations was expected to expedite the impact of soil abatement on the child's environment.

In the home, house dust is a mixture of street dust and soil dust, interior and exterior paint dust, workplace dust carried home by adults, and dust generated from human activities within the household. It is believed that most of the mass of the interior dust originates from soil immediately exterior to the home, but this can vary greatly by the types of family activities and by neighborhood characteristics. Nevertheless, in the absence of lead-based paint inside the home, it would seem reasonable to assume that most of the lead in household dust comes from soil and other sources immediately outside the home.

Many of the Boston and Baltimore households selected for the project had chipping and peeling lead-based paint, both interior and exterior. In order to reduce the impact of this paint, the walls and other surfaces were scraped and smoothed, then repainted. It is important to note that this approach is not a full scale paint abatement and was not designed

to permanently protect the child from lead-based paint. Paint stabilization was used on interior surfaces in Boston, and on exterior surfaces in Baltimore. Paint stabilization was not used in Cincinnati, because the lead-based paint was believed to have been removed from these homes in the early 1970s as part of a housing rehabilitation project.

In order to accurately measure the effectiveness and persistency achieved by soil abatement and the impact of this abatement on reducing lead exposure for children, the sampling and analysis plans for soil and dust required robust quality control and quality assurance objectives. Protocols were developed to define sampling schemes that characterize the expected exposure to soil for children; collect, transfer, and store samples without contamination; and analyze soil, dust, handwipe, and blood samples in a manner that would maximize interlaboratory comparison. The original design focussed on sampling blood lead during the late summer, as it was known that the seasonal blood lead cycle peaks during this time. Where this schedule could not be adhered to, an effort was made to schedule the follow-up blood lead sampling at a comparable time in the cycle.

Information on area treated and volume of soil removed from each of the three cities properties appears in Table 1-3. A total of 35 Boston properties were abated during the study. In Baltimore, 63 properties in the BAL SP treatment group (see Table 1-3) were abated between August and November 1990. An additional seven properties that did not meet the requirements for abatement were transferred to a control group. Unpaved surfaces were divided into areas on each property (usually front, back, and one side) and any area with the maximum soil lead concentration above 500 μg/g was abated entirely.

Within each of six neighborhoods, the Cincinnati study identified all sites with soil cover as discrete study sites. The decision to abate was based on soil lead concentrations for each parcel of land, and for the depth to which the lead had penetrated. Lead was measured at two depths, the top 2 cm and from 13 to 15 cm. If the average concentration of the top and bottom samples was greater than or equal to 500 μg/g, the soil was removed and replaced. If the average of the top samples exceeded 500 μg/g, but the average of the bottom samples was less than 500 μg/g, the soil was also abated. Ground cover was reestablished on abated soils and some unabated soils according to protocols described in the Cincinnati Report (appended).

TABLE 1-3. SOIL ABATEMENT STATISTICS FOR THE THREE STUDIES

	Boston	Baltimore	Cincinnati
Number of properties[a]	35	63	171
Surface area (m^2)	7,198	4,100[b]	12,089
Volume soil removed (m^3)	1,212	690	1,813
Surface area/property (m^2)	200	73	71
Volume soil/property (m^3)	34	11[b]	11

[a]Includes only properties abated during the study. Properties abated at the end of the study, where no further sampling was reported, are not included in this analysis, but are included in the individual study reports. In Cincinnati, a property is the location of the soil abatement, not the location of the child's residence.
[b]Surface area not provided by Baltimore report. This was calculated using Boston volume-to-surface ratio, which is equivalent to an average removal depth of 17 cm.

Exterior dust abatement was performed in the Cincinnati study only. The approach to this abatement was to clean all types of hard surfaces where dust might collect, using vacuum equipment that they tested and found to remove about 95% of the available dust on the area. The dust surface categories were streets, alleys, sidewalks, parking lots, steps, and porches.

Dust measurements were made in a manner that determined the lead concentration (micrograms of lead per gram of dust), the dust loading (milligrams of dust per square meter), and the lead loading (micrograms of lead per square meter) for the surface measured. This required that a dry vacuum sample be taken over a prescribed area, usually 0.25 to 0.50 m^2. It is important to note that dust abatement is not expected to cause an immediate change in the lead concentration on dust surfaces, only in the dust and lead loading.

Household dust was abated in the Boston and Cincinnati studies, but not in Baltimore. The BOS SPI and CIN SEI groups (see Table 1-1) received interior dust abatement at the same time as soil abatement, the BOS PI-S group received interior dust abatement without soil abatement, and the three CIN I-SE neighborhoods received interior dust abatement in the first year, followed by soil and exterior dust abatement in the second year.

In Boston, interior dust abatement was performed after loose paint stabilization. Hard surfaces (floors, woodwork, window wells, and some furniture) were vacuumed, as were soft surfaces such as rugs and upholstered furniture. Hard surfaces were also wiped following vacuuming. Common entries and stairways outside the apartment were not abated.

The Cincinnati group performed interior dust abatement after exterior dust abatement. Vacuuming was followed by wet wiping with a detergent. They vacuumed hard surfaces and replaced one to three carpets and two items of upholstered furniture per housing unit. Their previous studies had shown that these soft items could not be cleaned effectively with vacuuming alone.

Most homes in the Cincinnati group had undergone extensive rehabilitation, which was believed to have removed the lead-based paint 20 years prior to the project, but in Boston and Baltimore lead-based paint occurred in nearly every home. Because full paint abatement was not within the scope of this project, the alternative was to retard the rate of movement of lead from painted surfaces to household dust to the extent possible. The interior surfaces of all Boston homes and the exterior surfaces of all Baltimore homes received loose paint stabilization approximately one week before soil abatement.

In Boston, loose paint stabilization consisted of removing chipping and peeling paint and washing the surfaces. Window wells were painted with a fresh coat of primer. Baltimore homes were wet scraped over the chipping and peeling surfaces, followed by vacuuming. The entire surface was primed and painted with two coats of latex paint.

1.2 SUMMARY OF INDIVIDUAL STUDY REPORTS

Following the completion of data collection and analyses, the research teams in each city prepared individual study reports characterizing in detail the study design, procedures, and results obtained in their respective cities. Some of the more salient features of each study and key findings reported by the individual city investigators are summarized next.

1.2.1 Boston Study

The Boston study retained 149 of the original 152 children enrolled, although 22 children moved to a new location while continuing in the study. Children with blood lead concentrations below 7 μg/dL or above 24 μg/dL had been excluded from the study and two children were dropped from some aspects of the data analysis when they developed lead poisoning, probably due to exposure to lead-based paint abatement debris at a location away from their home.

Baseline characteristics (age, SES, soil lead, dust lead, drinking water lead, and paint lead) were similar for the three study groups (BOS P-S, BOS PI-S, BOS SPI). The preabatement average blood lead concentration was highest for BOS P-S. The proportion of Hispanics was higher in BOS P-S than in BOS PI-S or BOS SPI, and the proportion of blacks was lower. There was a larger proportion of male than female children in BOS P-S.

Data were analyzed by analysis of covariance (ANCOVA), which showed a significant effect of intervention for both the BOS PI-S and BOS SPI groups. These results did not change following adjustment for age, sex, SES, or any other variable except race and paint. When the paint variable was controlled, the blood lead declines were diminished and the results were borderline statistically significant. When the race variable was added, the blood lead declines were also diminished, and the results were not statistically significant.

Participants were chosen to be representative of the population of urban preschool children who are at risk of lead exposure. The Boston Childhood Lead Poisoning Prevention Program identified potential participants from neighborhoods with the highest rates of lead poisoning. Because study candidates with blood lead levels below 7 μg/dL or in excess of 24 μg/dL at baseline were excluded from the study, no conclusion about the effect of abating lead contaminated soil for children outside of this range can be made. Similarly, a different effect might have been found for children having a greater blood lead contribution from soil, such as in communities with smelters or other stationary sources where soil lead levels are substantially higher than those seen in this study or where differences in soil properties result in differences in bioavailability.

Follow-up blood lead measurements were made in Boston 11 months after intervention and again at 23 months.

1.2.2 Baltimore Study

The Baltimore study recruited 472 children, of whom 185 completed the study; and of those that completed the study, none were excluded from analysis. The recruited children were from two neighborhoods, originally intended to be a treatment and a control group. Because soil concentrations were lower than expected, some properties in the treatment group did not receive soil abatement. The Baltimore report transferred these properties to the

control group. In this report, the unabated properties in the treatment group are treated as a separate control group.

Because of logistical problems, there was an extended delay between recruitment and soil abatement that accounted for most of the attrition from the project. In their report, the Baltimore group applied several statistical models to the two populations to evaluate the potential bias from loss of participating children. These analyses showed that the two populations remained virtually identical in demographic, biological and environmental characteristics.

The Baltimore study provided limited information on the impact of house dust as a part of the change in lead in the child's environment. The study design focused on changes in biological parameters, hand dust and blood lead, over an extended period of time. There were no measurements of exterior dust, no interior paint stabilization, and no interior dust abatement. Except for the abated properties, there were no follow-up measurements of soil lead concentrations.

Including the prestudy screening measurements of hand dust and blood lead in the original cohort of participants, the Baltimore study included six rounds of biological measurements that spanned 20 months, including postabatement measurements made at 2, 7, and 10 months following abatement.

1.2.3 Cincinnati Study

The Cincinnati study recruited 307 children, including 16 children born to participating families during the study, and an additional 50 children who were recruited after the beginning of the study. In their primary data analysis, the Cincinnati group excluded these 66 children who were recruited after the start of the study, plus 31 children who were living in nonrehabilitated housing suspected of having lead-based paint and four children (in two families) who had become lead-poisoned from other causes. Thus, data for 206 children were analyzed in the Cincinnati report, and results for these 206 children are included in this integrated report along with 7 of the 31 children living in nonrehabilitated housing. The remaining 24 were dropped because of insufficient follow-up data.

The Cincinnati study abated soil on 140 parcels of land scattered throughout six neighborhoods. If soil were the only source of lead in the neighborhoods, exterior and

interior dust should have responded to the reduction in soil lead concentrations. However, exterior dust lead loading decreased only slightly following both soil and dust abatement, and returned to preabatement levels within one year. Corresponding changes in house dust, hand lead, and blood lead paralleled changes in exterior dust. Interior dust returned to preabatement levels about one year after abatement. Because blood lead concentrations also decreased in the control area, the Cincinnati group concluded that there is no evidence for the impact of soil and dust abatement on blood lead concentrations. However, this integrated report concludes, through a detailed structural equation analysis, that there is a strong relationship between entry dust and interior dust in this subset of the Cincinnati study, where the impact of lead-based paint was minimized.

Postabatement measurements in Cincinnati were made at 2, 10, 14, and 21 months following abatement in the first year, and at 3 and 10 months following abatement in the second year.

1.2.4 Individual Study Conclusions

In their individual city report following the first phase of their study, the Boston group stated their conclusions as follows:

- *"...this intervention study suggests that an average 1,856 ppm reduction in soil lead levels results in a 0.8-1.6 μg/dL reduction in the blood lead levels of urban children with multiple potential sources of exposure to lead."*

During Phase II of the Boston study, soil abatement was conducted in the two comparison groups (BOS PI-S and BOS P-S) and follow-up was extended another year in order to assess the generalizability and persistence of the blood lead decline observed in Phase I. Following the second phase of the study, the Boston group concluded (Aschengrau et al., 1994):

- *The blood lead "reduction in Phase II was somewhat greater than that in Phase I. The combined results from both phases suggest that a soil lead reduction of 2,060 ppm is associated with a 2.2 to 2.70 μg/dL decline in blood lead levels."*[3]

The basis for their conclusions consisted of an analysis of variance comparing mean blood lead changes among the three intervention groups, paired t-tests for within group

[3]This value for soil, 2,060 ppm, cited in their published report, was not adjusted by the Boston group with the interlaboratory correction factor of 1.037 in Table 3-6.

effects, and analysis of covariance with one-at-a-time adjustment for age, SES, race, sex, paint, water, and mouthing behavior. The analysis of covariance was performed using no transformation of blood lead data, which appeared to be normally distributed.

The Baltimore group stated their conclusions as follows:

- *"Statistical analysis of the data from the Baltimore Lead in Soil Project provides no evidence that the soil abatement has a direct impact on the blood lead level of children in the study."*

- *"In the presence of lead-based paint in the children's homes, abatement of soil lead alone provides no direct impact on the blood lead levels of children."*

The basis for these statements consisted of an adjusted and unadjusted analysis of selected covariates. The natural log of the blood lead of children in the treatment group showed no significant difference from the natural log of the blood lead of children in the control group, even when adjustments were made for age, SES, hand lead, season, dust, soil, sex, weak mouthing behavior, or strong mouthing behavior. These analyses were made on two sets of data. The first set consisted of all children enrolled in Rounds one and six. The second group consisted only of children enrolled in all six rounds.

The Cincinnati conclusions can be paraphrased from their report as follows:

- Following interior and exterior dust and soil lead abatement, blood lead concentrations decreased (in Area A) from 8.9 to 7.0 (21%) but increased to 8.7 μg/dL at 10 mo postabatement. Following interior dust abatement alone blood lead concentrations decreased from 10.6 to 9.2 (13%) 4 mo postabatement and were 18% below preabatement 10 mo postabatement. In the two neighborhoods with no abatement, blood lead levels decreased by 29 and 6% during these same time periods. Other comparisons also revealed no effects of the soil or dust abatement.

- There was no evidence that blood lead levels were reduced by soil lead or dust abatement in Area A (with soil, exterior dust, interior dust abatement). There was a slight reduction (net reduction over control area) of 0.6 μg/dL in Area B that might be attributed to interior dust abatement. This difference is not statistically significant.

The basis for the Cincinnati conclusions was a comparison of log transformed mean blood lead concentrations in the three treatment groups between Rounds 1 and 4.

1.3 SUMMARY OF EPA INTEGRATED ASSESSMENT RESULTS AND FINDINGS

The original data sets for each of the three participating cities were submitted to EPA, along with the individual study reports referred to above. Further analysis of the data were conducted by EPA staff in ORD, especially in the Environmental Criteria and Assessment Office/Research Triangle Park, NC (ECAO/RTP), now the National Center for Environmental Assessment (NCEA-RTP). The present intergrated report summarizes information on the additional EPA statistical analyses and their results.

From the perspective of the child's environment, changes in the soil lead concentration are expected to bring about changes in the house dust concentration, the hand dust, and the blood lead concentration. In each of the three studies, the soil lead concentrations were reduced to approximately 25 to 200 $\mu g/g$ in the study area, and for many treatment groups, there was a reduction of group mean blood leads, although not always statistically significant.

1.3.1 Quality of the Data

In the absence of certified standards for soil and dust, it was necessary to implement a program that would ensure that chemical analyses performed by the three participating laboratories would be internally accurate and externally consistent with similar analyses by other researchers. This program consisted of identifying acceptable analytical and instrumental methods, establishing a set of soil and dust standards, and monitoring the performance of the participating laboratories through an external audit program.

Chemical extraction of an estimated 75,000 soil and dust samples per study presented a costly burden for the project both in terms of time and expense and there were advantages associated with nondestructive analysis for a project of this nature. Because of these considerations, the Scientific Coordinating Panel recommended the use of laboratory scale X-ray fluorescence (XRF) for soil analysis, on the condition that a suitable set of common standards could be prepared for a broad concentration range and that a rigorous audit program be established to ensure continued analytical accuracy. Two groups, Boston and Baltimore, elected to use laboratory XRF for interior dust analysis also, whereas Cincinnati opted for hot nitric acid extraction with atomic absorption spectroscopy (AAS) for interior dust and XRF for exterior dust. During the study, the Baltimore group recognized problems

with analyzing dust by XRF when the sample size was small, less than 100 mg. They reanalyzed the dust samples by AAS and reported both measurements. In Boston, this problem was solved by compositing the floor dust samples for XRF analysis, reporting one floor dust sample per housing unit.

During the project, there were two rounds of soil and dust interlaboratory calibration exercises, one near the beginning and one at the completion of the soil and dust analyses. These exercises, which involved the three participating laboratories and two additional laboratories for each exercise, provided the basis for the evaluation of the performance of each laboratory in the audit sample program, and for the conversion factors used to compare soil and dust data between laboratories.

Each study maintained rigorous standards for database quality. These included double entry, 100% visual confirmation, and standard procedures for detecting outliers. Some errors were found during the preparation of this report, and they were corrected in consultation with the pertinent individual city investigators prior to use in this report. None of these errors would have impacted the conclusions drawn by the individual study.

1.3.2 Effectiveness and Persistency of Intervention

Soil abatement reduced soil concentrations in all three studies, and there was no evidence of soil recontamination in either Boston or Cincinnati. There were no follow-up measures of soil in Baltimore that would detect recontamination. There was some evidence for exterior dust recontamination in Cincinnati. The Cincinnati group suggests that this might be caused by chipping and peeling lead-based paint from the exterior surfaces of nearby buildings not included in the project.

Interior dust abatement was persistent in both Boston and Cincinnati, even though some recontamination occurred in Cincinnati in response to the exterior dust recontamination. Paint stabilization appeared to have some impact on exposure, but there were no measures of persistency.

1.3.3 Comparison of EPA Integrated Report Results with Individual Study Results

This integrated assessment looks at the three individual studies collectively to determine if a broad overview can be taken of the project results when each study is placed in its correct perspective.

1.3.3.1 Boston Study

The key findings of this integrated assessment with regard to the Boston study are as follows:

1. The median preabatement concentration of lead in soil was relatively high in Boston, averaging about 2,400 μg/g with few samples below 1,000 μg/g.

2. Abatement of the soil effectively reduced the median concentration of lead in the soil to about 150 μg/g (an average decrease of about 2,300 μg/g).

3. Soil was clearly a part of the exposure pathway to the child, contributing significantly to house dust lead.

4. Other sources of lead, such as interior lead-based paint were minimized by stabilization.

5. The reductions of lead in both soil and house dust persisted for at least two years.

6. Blood lead levels were reduced by approximately 1.86 μg/dL at 10 mo after soil lead abatement.

7. Additional reductions in blood lead of about 2.0 μg/dL (relative to non-abated) were observed at 22 mo postabatement for children in houses where the soil lead was abated and the interior house dust lead was consequently reduced and remained low.

The Boston study used analysis of variance methods based on blood lead differences, and analysis of covariance methods with the longitudinal aspect included by use of the pre-abatement blood lead concentration (Round 1) as a covariate. The results of their "crude" analysis (Table 15-10 in the Boston study report) are virtually identical to the effect size estimates calculated by U.S. EPA for the group as a whole using repeated measures ANOVA and also using a longitudinal structural equations model. The results are shown in Table 1-4. The effect size estimates are somewhat smaller in their "base" model, which the

TABLE 1-4. COMPARISON OF PHASE 1 EFFECT SIZE ESTIMATES BETWEEN THE BOSTON STUDY AND THIS REPORT

GROUP STUDY		BOSTON REPORT[1]		THIS REPORT[1]	
ABATE VS CONTROL		CRUDE MODEL	BASE MODEL	RM ANOVA	LSEM MODEL 17
BOS SPI	BOS P-S	1.92	1.49	1.87	1.86
BOS SPI	BOS PI-S	1.53	1.28	1.54	1.56
BOS PI-S	BOS P-S	0.39	0.21	0.33	0.30

[1] Units are μg/dL reduction of Pb in blood.

longitudinal analysis of covariance model adjusted only for pre-abatement blood lead. In view of the differences in methods and approaches, the overall conclusions are very similar.

The Boston investigators also studied the sensitivity of the effect size estimates to a large number of other covariates, including environmental factors, family demographic factors, behavioral factors, and biological covariates. None of these changed the estimated effect of BOS SPI versus BOS P-S (soil abatement versus control) from their base model, 1.49 μg/dL, by more than 0.22 μg/dL. The factors were entered one at a time. The largest decrease was by inclusion of race as a factor, which reduced the effect to 1.27 μg/dL, and by inclusion of pre-abatement lead paint, which reduced the estimated effect to 1.34 μg/dL. Five factors decreased the effect size, which nevertheless remained statistically significant: water lead concentration, time away from home, time away from study area, playing or sitting on inside floor, and ferritin level. The other 15 factors tested increased the estimated effect size, particularly age (to 1.61 μg/dL) and hand washing before meals (to 1.63 μg/dL), as well as: gender, socioeconomic status, mouthing variables, chipping paint, yard play, outdoor eating, hand washing after outdoor activity, pets that go outdoors, imported canned food, lead-related occupations, lead-related hobbies, cigarette smoking, and owner occupancy. Many of these factors are important in identifying individual exposure components and lead risk factors and are worthy of additional scientific investigation. However, none of these factors appear to have interacted so strongly with soil and dust abatement as to have qualitatively affected the conclusions of the study, except for relatively small effects related to age, race, and lead paint level. Much of the lead paint effect is

mediated, both statistically and physically, by lead concentrations or loadings in house dust. It is likely that the use of household dust as a covariate in the models of this report effectively subsumed the lead paint effect, and that the dust abatement carried out in the Boston study along with soil abatement may have affected some fraction of the blood lead response that might have been otherwise attributed to lead-based paint. Even so, the overall treatment group effect in the model that included lead paint was only slightly less significant ($P = 0.05$) than the base model ($P = 0.02$). Conversely, including chipping paint in the model increased the effect to 1.53 μg/dL ($P = 0.02$ for the group model, $P = 0.01$ for the BOS SPI versus BOS P-S effect). Additional studies involving the paint contribution to the total lead exposure pathways, and assessment of the possible effects and interaction between paint condition and paint lead loading on lead exposure, are needed to understand the relatively small modifications of effect size attributable to lead paint.

Age and race effects are larger than the paint effects, and were evaluated in this report. Larger effects were identified for children of ages 18 to 41 months, and for children of Afro-American ancestry, than for the sample as a whole. The Afro-American children also seemed to show larger responses to dust abatement than did the sample as a whole.

In summary, the abatement of soil in the Boston study resulted in a measureable, statistically significant decline in blood lead concentrations in children, and this decline continued for at least two years. It appears that the following conditions were present, and perhaps necessary for this effect: (a) a notably elevated starting soil lead concentration (e.g., in excess of 1,000 to 2,000 μg/g); (b) a marked reduction of more than 1,000 μg/g in soil lead consequent to soil abatement accompanied by (c) a parallel marked and persisting decrease in house dust lead.

These conclusions are consistent with those reported by the Boston research team. This integrated assessment found no basis for modifying their conclusions, although we choose not to express these findings as a broadly generalizeable linear relationship between soil and blood, such as change in micrograms of lead per deciliter of blood per change in micrograms of lead per gram of soil, because we believe that such a linear expression of abatement effects is highly site specific for the soil-to-blood relationship. We found evidence that the dust-to-blood relationship is more significant than the soil-to-blood relationship and therefore the abatement effect also depends on soil-to-dust transfer, which may be very site-specific.

1.3.3.2 Baltimore Study

With regard to analyses of Baltimore data conducted for this integrated assessment, the participants in the abatement neighborhood that did not receive abatement were treated as a separate control group, rather than being combined with the nonabatement neighborhood (as the Baltimore research team did). The reason for this was to establish a control group not influenced by differences between neighborhoods. This alternative approach used in this integrated assessment had little impact on the statistical significance of soil abatement effects as reported by the Baltimore research team.

The key findings of this integrated assessment for Baltimore are:

1. The preabatement concentrations of lead in soil were notably lower (i.e., averaging around 500 to 700 μg/g, with few over 1,000 μg/g) than in Boston.

2. The actual reduction of lead in soil by abatement was small (a change of about 400 μg/g), compared to the Boston study (a change of about 2,300 μg/g).

3. Measurements of blood lead were made for only ten months following abatement; and no significant decreases in blood lead consequent to soil abatement were observed compared to non-abatement control group children.

4. Except for exterior lead-based paint, there was no control of other sources of lead, such as the stabilization of interior lead-based paint (as done in Boston) or abatement of house dust (as done in Boston and Cincinnati).

5. Follow-up measurements of soil (except immediately postabatement) were not made to establish the persistency of soil abatement, and its possible effects on house dust.

The Baltimore report used a generalized linear regression model (GLIM). In its simplest form, the regression model can be expressed as a linear model using log-transformed variables. The Baltimore blood lead model 1 is a simple ANOVA model,

$$\text{Log}(BC_{ij}) = G_{gj} + e_{ij}$$

with only two treatment groups, Area 1 and Area 2. However, Area 1 includes some non-abated residences as well the residences that received soil abatement, whereas Area 2 includes only non-abated residences. Therefore, the results in the Baltimore report cannot be directly compared with the results reported here, where we have separated the abated and

non-abated residences into two groups and used the non-abated residences in Area 1 as a second control group. Model 2 in the Baltimore report is a simple ANCOVA model,

$$\log(BC_{ij}) = G_{gj} + b_{2j}\,Age_{ij} + b_{3j}\,SES_i + b_{4j}\,Season_{ij} + b_{6j}\,M_{ij}\log(Hand_{ij}) + b_{7j}\,(1-M_{ij})\log(Hand_{ij}) + e_{ij}.$$

In this notation, Age is a semi-categorical variable, Season is included only for pre-abatement rounds 1 and 2 that covered many months, and M_{ij} is a dummy variable for low or high mouthing behavior. While temporal comparisons are possible, no temporal correlation model is assumed, and the Baltimore report notes that the lack of temporal modeling is a deficiency in the analyses.

The Baltimore analyses were carried out for two distinct subgroups of children. The first set of analyses used only those children who were present in all six rounds. The second set of analyses used all children who were present in each round. Analyses for this EPA Integrated Report used children who were present in Rounds 3, 4, and 6. The set of children who were present in all rounds is included in the EPA set, but does not include other children in the EPA set (such as those children who were recruited at Round 3, especially very young children). The second set of children in the Baltimore study is much closer to the EPA children set in Rounds 4 and 6, but includes in Round 3 some additional children who dropped out after Round 3. Therefore, the EPA effects size estimates are based on somewhat different groups of specific children than in the Baltimore report.

Effect sizes were calculated in Table 1-5 as simple differences of treatment group effects reported in Tables 7-7 and 7-8 of the Baltimore report. The effects were small and probably not statistically significant, although the lack of correlation structure in the Baltimore models makes any estimates of standard errors rather questionable. The differences in blood lead are negative between the treatment group (BAL SP) and the control group (BAL P1 and BAL P2). There is little reason to believe that major treatment group differences would have been identified by other analyses of these data.

Other findings in the Baltimore study are of interest. There were some indications of significant differences associated with hand lead, with a modifying effect due to child mouthing behaviors. There was also a strong effect of socioeconomic status on blood lead

TABLE 1-5. EFFECT SIZE ESTIMATES FROM THE BALTIMORE REPORT COMPARING BLOOD LEAD REDUCTION IN BAL SP VERSUS CONTROLS

		BALTIMORE MODEL[1,2]		THIS REPORT[1,3]	
ROUNDS	CHILD GROUP	ANOVA	ANCOVA	BAL SP vs BAL P1	BAL SP vs BAL P2
ROUNDS 3 AND 4	ALL 6 ROUNDS	-0.55	0.12	0.07	1.77[4]
	EACH ROUND	-0.07	-0.10		
ROUNDS 3 AND 6	ALL 6 ROUNDS	-0.92	-0.71	-0.54	0.67
	EACH ROUND	-1.55	-1.17		

[1] Units are μg/dL reduction of Pb in blood.
[2] Baltimore controls are BAL P1 and BAL P2.
[3] Children present in Rounds 3,4, and 6.
[4] P=0.16; others, P>0.2.

and dust lead, and an age effect with maximum blood leads at ages 1 to 3 years (12 to 36 months), a general finding in these studies.

Thus, in Baltimore, where the difference between pre- and postabatement soil lead concentrations was much less than in Boston, and where the soil abatement criteria left some properties only partially abated and no interior paint stabilization or dust abatement was performed, no detectable effects of soil lead abatement on blood lead levels were found.

These conclusions are consistent with those reported by the Baltimore research group, and are not inconsistent with those above for the Boston study. At soil concentrations much lower than the Boston study, the Baltimore group would have likely been able to see only a very modest change in blood lead concentrations (perhaps less than 0.2 μg/dL) assuming similarity between the study groups in Boston and Baltimore and the same linear relationship between change in soil concentration and change in blood lead. Furthermore, the interior paint stabilization and house dust abatement performed in Boston likely enhanced and reinforced the impact of soil abatement on childhood blood lead, whereas in Baltimore, any

possible small impact of soil abatement would have likely been swamped by the large reservoir of lead in the interior paint and the large unabated amounts of lead in interior house dust.

1.3.3.3 Cincinnati Study

As for the Cincinnati study, because of differences in the neighborhoods, we found that combining neighborhoods into treatment groups often obscures important effects; and so we chose to analyze each of the six Cincinnati neighborhoods as separate treatment groups. One neighborhood, Back Street, had an insufficient number of participants and was dropped from some analyses; that group started with nine families, but by Round 5 there was only one participating family in the study. We also found that the two control neighborhoods, Glencoe and Mohawk, were substantially different, and that the three remaining treatment groups (Pendleton, Dandridge, and Findlay) were more comparable, both demographically and in geographic proximity, to Mohawk than to Glencoe.

The Cincinnati study used several different regression (ANCOVA) models, and cross-sectional structural equation models. Their individual city report also included results of a simple correlation analysis that did not allow for multiple covariate adjustments, and is not further described. The response variables in the regression models included differences in blood lead between Round 1 and Round 4, hand lead differences, and differences in interior floor dust loading and in exterior dust loading. The final regression model for the change in blood lead involved only blood lead concentration (which we denote Blood), hand lead loading (which we denote Hand), age of the child at the Round 4 blood lead measurement (which we denote $Blood_{i4}$), and socioeconomic status (denoted SES). In our notation, their model is:

$$Blood_{i4} - Blood_{i1} = 8.52 + 0.038\,(Hand_{i4} - Hand_{i1}) - 0.00079\,Age_{i4}{*}Hand_{i4} - 0.17\,SES - 0.43\,Blood_{i1}.$$

This model has one point of similarity to our Cincinnati longitudinal SEM models. By transposing the $Blood_{i1}$ on the left side of the equation, we have a linear relation that is expressed algebraically as $Blood_{i4} = 8.52 + \ldots$ other terms $+ 0.57\,Blood_{i1}$, which is close

to the value of the blood lead persistence parameter A_{14} obtained for most of the Cincinnati LSEM models, such as $A_{14} = 0.58$ in Model J6 used in the effects size comparisons. Otherwise, blood lead is not predicted by neighborhood, nor by abatement group, nor by environmental lead concentrations or loadings, but by another time-variable and child-specific variable, hand wipe lead loading, which tends to increase with the child's age. The regression model for hand lead change also excludes treatment group or environmental variables, except indirectly through Round 1 hand lead.

Their report also presents a structural equation model for blood lead and hand lead differences, and for changes in interior and exterior dust lead. Their equations for blood and hand lead are, in our notation:

$$Blood_{i4} - Blood_{i1} = 10.28 - 0.18\ SES - 0.064\ Age_{i5} - 0.46\ Blood_{i1}$$

$$Hand_{i4} - Hand_{i1} = 5.78 + 0.002\ Hand_{i5} - 0.62\ Hand_{i1}.$$

The two dust lead equations are totally unconnected to blood lead or hand lead.

The Cincinnati report also shows cross-sectional structural equation models for Round 1, Round 3, and Round 4 respectively. The Round 1 SEM model shows large and statistically significant age effects, and effects of mouthing behavior. Areas and neighborhoods show no significant differences. The Cincinnati cross-sectional SEM model uses no environmental covariates, but reports a significant regression of log(BloodR1) on log(HandR1). The simultaneous equation for log(HandR1) depends strongly on age and not at all on treatment group or neighborhood. Neither equation uses any of the environmental covariates, but both include a significant fixed effects factor for "families", which is analogous to the random effects term Hh(g) in our EPA repeated measures ANOVA and ANCOVA models. However, their findings of no significant neighborhood differences or environmental factors differs somewhat from some of the findings in our EPA cross-sectional and longitudinal SEM models. Differences in model format and structure make direct comparisons very difficult.

The Cincinnati investigators concluded that the Phase 1 changes in blood lead concentrations and in hand lead loadings were not significantly different among the three

abatement groups, using either multiple regression models or structural equation models. They did not compare across different neighborhoods within treatment groups, which was an additional source of variability in the study. We cannot therefore directly compare our effect sizes or treatment differences across neighborhoods with their aggregated results. Since their models are not directly comparable to our models without additional substantive analyses of the role of hand wipe lead, we cannot directly compare effect sizes using longitudinal SEM.

The Cincinnati report giving a cross-sectional SEM for Round 4 (their Table 4-63) presents a comprehensive and detailed SEM, which is in substantial qualitative agreement with the EPA longitudinal SEM presented here for Cincinnati Round 4 blood lead and dust lead. The use of hand lead in their model precludes direct comparisons with the longitudinal SEM in Table 5-39. The use of log(HandR4) as a covariate that is only partially adjusted by window and floor dust lead loadings, age, and SES permits the finding of large, statistically significant, but *negative* relationships between log(BloodR4) and dust lead loadings on the floor, interior entry, and exterior. Additional analyses of this model would be useful. The model uses neighborhood or area as an adjustment covariate for hand-to-blood, dust-to-blood, dust-to-hand, paint-to-dust, and exterior-to-floor pathways, with some significant differences. While the application of this model does not allow comparison of effect sizes relative to Round 1, there is a qualitative similarity between our EPA findings and those of the Cincinnati investigators.

On this basis, we concluded that, in most cases, the effect of soil abatement could not be clearly determined, and offer the following explanation for this conclusion:

1. Most of the soil parcels in each neighborhood were not adjacent to the living units, and this soil was therefore not the primary source of lead in house dust. Evidence for this statement includes the observation that street dust lead concentrations are much higher than soil concentrations, indicating there is a large source of lead contributing to street dust in addition to soil lead.

2. The preabatement median soil lead concentrations in the three treatment groups were about 300 μg/g in Pendleton, 700 μg/g in Findlay, and 800 μg/g in Dandridge, and the postabatement soil concentrations were less than 100 μg/g, so that the reduction of lead in soil was small, as in Baltimore.

Evidence for the impact of dust abatement or dust and soil abatement consists of a statistically significant difference between changes in blood lead between Rounds 1 and 4, approximately one year apart. Some Cincinnati neighborhoods showed decreased blood lead

concentrations in response to dust abatement or dust and soil abatement. The two neighborhoods that received only interior dust abatement in the first year, Dandridge and Findlay, showed a small decrease in blood lead concentrations, compared to large increases in the nearest control group, Mohawk. The treatment group that received soil, exterior dust and interior dust abatement, Pendleton, showed a smaller effect than did the Dandridge and Findlay neighborhoods. After consultation with the Cincinnati research team, we suspect that there was recontamination of street dust in Pendleton during the study, probably caused by demolition of nearby buildings in the neighborhood.

The consistent theme across the outcomes for all three studies is that soil abatement must effectively reduce soil lead concentrations for an extended period of time and be accompanied by a corresponding reduction in house dust lead in order to result in any detectable reduction of blood lead. The location of the soil relative to the exposure environment of the child is important. In this project, the movement of lead from soil or street dust into the home seems to be a key factor in determining blood lead concentrations. Although these USLADP results provide substantial evidence for the link between soil or street dust and house dust lead, there is insufficient information by which to clearly quantify this relationship in terms of the lowest level of soil or street dust lead reduction that will yield a measurable decrease of lead in blood.

1.3.3.4 Synthesis of Findings Across the Three Studies

While the USLADP was not intended to compare different methods for soil abatement, the differences in design and methodology among the three studies helped to identify conditions for which soil abatement may be an effective intervention, and conditions under which soil abatement is less likely to be effective. Abatement or intervention can be effective if it can achieve one or both of the following goals:

1. Abatement or intervention produces an effective and persistent reduction in the concentrations of lead in soil and in household dust.

2. Abatement or intervention changes childhood lead exposure by reducing the intake of lead-contaminated media, or effectively breaks the transport pathway from the lead-contaminated source to the child's activity areas.

These are not mutually exclusive goals, but there are important distinctions between them. The first goal, reducing lead concentrations, can be achieved without changing exposure or transport. For example, removing bare lead-contaminated soil from a yard and replacing it with bare soil that is not contaminated will not change the child's exposure nor the transport of surface soil from the yard into the house. However, the child's intake of lead from soil ingestion will immediately be reduced, and one would expect that over some period of time, there will be a reduction of the child's intake of lead from household dust because the soil component of household dust lead has been eliminated. All three studies achieved the elimination of lead in yard soil. It is important to note the requirement that the soil not be recontaminated by unremediated sources such as exterior paint and by transport of lead from unremediated areas. Even in the Boston study, a few soil-abated yards became substantially recontaminated. However, most of the sampled locations in the Boston and Cincinnati did not suffer significantly recontaminated soil after abatement. The Baltimore sites were not followed up over a similar period of time.

Both Boston and Cincinnati residences received interior dust abatement. The Boston residences showed slight evidence of recontamination, whereas most the residences in the Cincinnati areas that received interior dust abatement (with or without soil abatement) during Phase 1 of the study showed significant recontamination. The floor dust lead concentrations showed a significant association with window lead and mat lead, suggesting exterior sources of recontamination. Long-term changes in dust lead were not followed up in the Baltimore study. Significant blood lead reduction was detected only in the Boston study, where persistent reduction of dust lead occurred in most residences that received soil lead and interior dust abatement. The effect was even greater in Phase 2 in the group BOS PI-S that received both Phase 1 dust abatement and both soil and dust abatement in Phase 2.

The second goal, reduction of exposure, requires reducing the amount of potentially lead-contaminated media consumed by the child. The Boston study shows some indication that this also may have occurred, whereas the Cincinnati provides little indication of

exposure reduction. Soil abatement can reduce exposure by covering soil with sod or other barriers that reduce the child's access to surface soil particles. The reduction in exposure is distinct from reducing the lead concentration in the soil to which the child is exposed. Likewise, frequent and effective washing or vacuuming of household dust can reduce the amount of dust (dust loading) that is accessible to the child, however much lead is in the dust. Changes in behavior, such as more frequent hand washing or greater parental attention, can also reduce contact with dust and soil. Since all of these studies may have initiated behavioral changes from the moment of recruitment simply by informing parents and caretakers of potential lead hazards, such changes cannot be detected with this study design.

The second goal can also be achieved by any process that reduces transport of the contaminant from the source to the areas in which the child may come into contact with it. Covering bare soil with sod, concrete, or other barriers will clearly prevent contamination of house dust and outside play areas, as the encapsulation of paint will prevent paint chips from contaminating dust, so long as the barrier remains intact. Removing the source of lead contamination was shown to be effective in Boston, but in addition to this, there is also some possibility that the post-abatement pathway regression coefficient from soil to dust may have been changed. However, there may also have been a serious attenuation of the apparent pathway in the Round 1 data set, possibly attributable to the blood lead truncation of the study. Additional studies on the effects of soil abatement on environmental lead pathway kinetics would be useful. In general, any method that attempts to estimate post-intervention or post-abatement blood lead concentrations (for example, EPA's IEUBK Model or "slope factor" models) should take into account not only the changes in environmental lead concentrations that may occur as a results of abatement or intervention, but also the changes in the pathways to childhood exposure that may occur following abatement or intervention.

Finally, one should recognize that any environmental lead abatement or intervention may be limited in its ability to reduce blood lead concentrations in currently lead-burdened children. It appears that in the first year after abatement, at most 40 to 50 percent of the child's existing blood lead burden may be removable by soil abatement or any other combination of abatements and interventions apart from medical treatment by chelation. There may be a much greater effect of lead abatement in preventing lead exposure for future

residents. Long-term benefits of lead abatement should therefore be considered in assessing abatement effectiveness, as well as short-term benefits.

1.4 INTEGRATED PROJECT CONCLUSIONS

The main conclusions of this Integrated Report report are two-fold:

(1) *When soil is a significant source of lead in the child's environment, under certain conditions, the abatement of that soil will result in a reduction in exposure that will cause a reduction in childhood blood lead concentrations.*

(2) *Although these conditions for a reduction in blood are not fully understood, it is likely that five factors are important in determining the magnitude of any possible reduction: (1) the past history of exposure of the child to lead, as reflected in the preabatement blood lead; (2) the initial soil lead concentration and the magnitude of the reduction in soil lead concentrations; (3) the initial interior house dust lead loading and the magnitude of reduction in house dust lead loading; (4) the magnitude of other sources of lead exposure, relative to soil; and (5) the strength of the exposure pathway between soil and the child relative to other lead exposure pathways in the child's environment.*

The basis for the first conclusion is: in Boston, where the soil lead concentrations were high (mostly > 1000 to 2000 $\mu g/g$) and the contribution from lead-based paint was reduced by paint stabilization, there was a measurable reduction of blood lead concentrations. This reduction continued to increase for two years following abatement in Boston.

Conversely, in Baltimore and Cincinnati, where soil was not a significant source of lead relative to other sources, there was no measurable reduction of blood lead except in cases where other sources were also removed or abated. In Baltimore, these sources may have been interior lead-based paint that was not stabilized, or house dust that was not abated. In Cincinnati, the principal source of lead seemed to be neighborhood dust that may have been contaminated with lead-based paint.

The basis for the second conclusion is: firstly, in those cases where all important elements of the exposure pathway were available for assessment, the structural equation model analyses showed that preabatement blood lead concentration was an important predictor of postabatement blood lead, suggesting that the remobilization of bone lead is an important component of the measured blood lead. Secondly, all other factors being equal, the measurable reduction in blood lead was observed only at higher concentrations of soil

lead. In the absence of information about other sources of lead, no clear statement can be made about the possibility of smaller reductions in blood lead at lower soil lead concentrations.

In spite of the recent successes in reducing exposure to lead by removing lead from gasoline and canned food, lead exposure remains a complex issue. This integrated assessment attempts to assess exposure to lead in soil and house dust. Lead in soil and lead-based paint are closely linked in the child's environment. If there is exterior lead-based paint, then soil lead is likely to be elevated with a consequent elevation in house dust lead. If there is interior lead-based paint, then efforts to reduce the impact of soil lead on house dust will be only partially effective. The maximum reduction in lead exposure will not be achieved unless both paint and soil abatement are implemented.

There is evidence from all three studies that lead moves through the child's environment. This means that lead in soil contributes to lead in street or playground dust, lead in exterior paint contributes to lead in soil, and lead in street dust contributes to lead in house dust. A more detailed analysis of the data may show the relative contribution from two or more sources, but the present analyses imply that this transfer takes place.

The analysis of the data from the three studies showed evidence that blood lead responds to changes in house dust lead. There is also evidence for the continued impact of other, independent sources following abatement of one source. This means that abatement of soil or exterior paint does not necessarily reduce the contribution of lead from other sources such as interior lead-based paint.

The conclusions of this report suggest that soil abatement can have a measurable effect on reducing exposure to lead if there is a substantial amount of lead in soil and if this soil lead is the primary source of lead in house dust. In such cases, both soil abatement and interior dust removal should be performed to be fully effective. Likewise, soil abatement should be considered in conjunction with paint abatement when it is likely that soil will otherwise continue to contaminate house dust after a paint abatement is completed.

From one perspective, decisions about soil abatement should be made on an individual home basis. This report shows that, on an individual house basis, soil abatement may reduce the movement of lead into the home and its incorporation into house dust. The magnitude of this reduction depends on the concentration of lead in the soil, the amount of soil-derived

dust that moves into the home, the frequency of cleaning in the home and the cleanability of the home. The number and ages of children and the presence of indoor/outdoor pets are factors known to increase this rate of dust movement, whereas frequent cleaning with an effective vacuum cleaner, use of entry dust mats, and removing shoes at the door serve to reduce the impact of soil lead on house dust.

From another perspective, soil abatement at the neighborhood level poses problems not pertinent to individual homes. Playground, vacant lot, and other plots of soil may pose an immediate problem if they are accessible to children and there is a direct pathway for dust generated by this soil to enter the home. Likewise, sources of lead other than soil may contribute more to exterior dust than soil itself. The evidence in this report suggests that the key to reducing lead exposure at the neighborhood level is to abate significant sources of lead contributing to exterior dust, in addition to the soil and paint abatement that would be performed on an individual property.

2. BACKGROUND AND OVERVIEW OF PROJECT

2.1 PROJECT BACKGROUND

2.1.1 The Urban Lead Problem

Children are exposed to lead through complex pathways from multiple sources. In the mid 1980s, when there was a dramatic increase in public concern for childhood lead exposure, attention focused on urban environments with high concentrations of lead in soil, where there was an apparent correlation with the incidence of high blood lead concentrations. At that time, there were several other sources of exposure that could potentially account for unusually high blood lead in a population of urban children. Among these were lead in the air (primarily from automobile emissions), lead in food (primarily from canned foods with lead soldered side seams), lead in drinking water (primarily from lead pipes or newly soldered copper pipes), and lead in paint. The lead in the soil was believed to be a mixture of lead from the atmosphere and lead from exterior paint. Regulations were in place that would largely remove lead from gasoline by the end of 1986, and there was a voluntary program among food processors to phase out cans with lead soldered side seams. Renewed public interest in paint abatement emerged in the late 1980's.

Prior to the start of this project, soil abatement had been performed in many nonurban residential areas with elevated soil lead. The decision to abate soil was usually based in part on the distribution of blood lead within the population of children. There was limited experience on the effectiveness of this abatement and little or no opportunity for follow-up studies of the results. There were little data from controlled evaluations because the intent of abatement was remediation, not experimentation.

2.1.2 Legislative Background

In the mid 1980s, the scientific evidence for a correlation between soil lead and blood lead was sufficient to warrant concern for the health of children, but not strong enough to support a large scale program for soil lead abatement. Consequently, the Urban Soil Lead Abatement Demonstration Project (USLADP), known also as the Three City Study, was

authorized in 1986 under Section 111(b)(6) of the Superfund Amendments and Reauthorization Act (SARA). SARA called for EPA to conduct a "pilot program for the removal, decontamination, or other actions with respect to lead-contaminated soil in one to three different metropolitan areas."

Although not specified in the amendment, the legislative history focused on lead-based paint as the source of lead in soil in urban residential areas. In response to the Superfund mandate, USLADP was designed to evaluate the effectiveness of removal of lead-contaminated soil in urban residential areas as a means to reduce blood lead levels of young, preschool children residing in abated residences or neighborhoods. The project was not designed to evaluate the relative effectiveness of different soil abatement technologies per se, but rather to focus on determining the extent to which the blood lead levels of children less than six years old (as a key risk group for lead health effects) could be reduced by intervention to decrease soil lead concentrations.

The EPA's Office of Solid Waste and Emergency Response (OSWER) had the principal responsibility for overall implementation of the project, as a Superfund-mandated activity. Administrative and financial management responsibilities, it was decided, were to be delegated to EPA regional offices for the geographic areas containing those cities selected for inclusion in the project. EPA's Office of Research and Development was asked to provide technical oversight and coordination assistance to help integrate scientific activities across the cities selected. An EPA Steering Committee was set up to oversee site selection and initiation of the project.

In 1987, EPA convened a set of experts to advise on the design of the project and to develop selection criteria for study sites. Six cities submitted proposals, and Boston, Baltimore, and Cincinnati were chosen by the following site selection process.

2.1.3 Site Selection

The three cities were selected based on an evaluation of each proposal in relationship to the following site selection criteria, as recommended by the experts.

A. To be considered for selection, a metropolitan area *must* have:

1. Agreement by the appropriate EPA regional office to provide general project oversight, and to disburse the funds.

2. An established entity, preferably the state, documented as willing to be responsible for removing and disposing of lead contaminated soil. This included identification of an appropriate facility within the state for disposal of the soil, facilitation of permits, community relations and education, and any other activities necessary to expeditiously provide for safe disposal.

3. The administrative infrastructure to carry out a large scale project. This included a key government department with appropriate authority to coordinate the project, and generally included active participation by the state, by community groups, and by all the different metropolitan departments with some responsibility for the project.

4. Access to scientific and medical expertise to ensure that sampling and analysis were properly conducted, and access to medical care needed for any children found to have lead toxicity.

5. Evidence that there are children with elevated blood lead levels (25 μg/dL as defined by the CDC in its 1985 childhood lead screening guidelines), and soil in residential areas with lead levels of 1,500 μg/g or greater.[1] It would be desirable for lead-based paint to be established as a major contributor to the soil lead levels.

B. To be considered for selection, a metropolitan area *should* have:

6. A documented high incidence of children with elevated blood lead levels in the proposed study areas. This meant that the municipality supported an active childhood lead screening program.

7. A pattern of high density population in study areas. The number of children available for evaluation as part of the project was important to the statistical validity of the study.

8. Availability of other sources of funding for portions of the project not funded by SARA. Such items might include de-leading the outside of houses, or intensive interior vacuuming to remove residual leaded dust.

The Steering Committee reviewed proposals from six metropolitan areas: Boston, Baltimore, Cincinnati, Minneapolis, Detroit, and East St. Louis. These were reviewed on December 3 and 4, 1987, by the Steering Committee and the set of expert consultants. Boston, Baltimore, and Cincinnati were selected based on the following key points:

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[1] Note that the stipulated soil value of 1,500 μg/g was interpreted as a significant number of soil parcels in which at least one soil measurement exceeded this value. Reports in this document of means or median values below 1,500 μg/g for individual soil parcels or entire treatment groups should not be misinterpreted as failure to meet the original selection criteria.

1. The Boston investigators proposed to select three groups of families randomly from several neighborhoods known to have soil lead concentrations in the range of 2000 to 5000 μg/g. One of these groups would receive only paint stabilization; a second group would receive paint stabilization and dust abatement, and the third group would receive soil abatement, dust abatement, and paint stabilization.

2. The Boston proposal involved collaboration among Boston City Hospital, Boston University, and the EPA Region I Laboratory (for conduct of analysis of lead in soil, dust, etc.). This collaborative group also had demonstrated experience with collection, analysis, and assessment of soil and blood lead data in inner city neighborhoods of Boston.

3. Cincinnati proposed a neighborhood level abatement study where housing units had been previously gutted and rehabilitated approximately 20 years ago, and most of the lead-based paint was either removed or encapsulated. The Cincinnati sites contained soil lead from 220 to 900 μg/g, exterior surface dust (primarily from paved areas) averaging 2,000 to 5,000 μg/g, and a number of children with blood lead concentrations above 25 μg/dL.

4. The Cincinnati proposal was prepared by the University of Cincinnati and demonstrated a high degree of organizational infrastructure, with commitments from the City of Cincinnati. There was an established infrastructure of neighborhood associations that was perceived to be a plus for the project.

5. The Baltimore project proposed individual housing units with soil lead concentrations averaging in excess of 1,000 μg/g. Lead-based paint had been abated in some, but not all houses.

6. The Baltimore proposal was prepared by the State of Maryland and showed a satisfactory level of organizational infrastructure and local scientific expertise; problems with the proposed statistical approach were resolved by consultation with the Steering Committee.

With the selection of Boston, Cincinnati, and Baltimore, a Scientific Coordinating Committee was established to provide scientific and technical support for the three studies and to coordinate the exchange of scientific information. The Steering Committee also recognized that there would be much value in standardizing and coordinating methods for media sampling and analysis. The Scientific Coordinating Committee was composed of representatives from the research teams of each of the three cities, the three EPA regional offices (Regions I, III, and V), the Office of Solid Waste and Emergency Response, the Environmental Criteria and Assessment Office/Research Triangle Park, NC (ECAO-RTP) (now the National Center for Environmental Assessment/RTP), and the Centers for Disease

Control and Prevention. The task of organizing, scheduling, and conduct of meetings of the Scientific Coordinating Committee was assigned to ECAO/RTP. Major policy decisions remained with the Steering Committee.

The funding mechanisms were set into place individually through the respective EPA regional offices (Regions I, III, and V). Each of these regional offices set up an independent funding mechanism and oversight plan. The regional project officer became the liaison to the Steering Committee and to the Scientific Coordinating Committee. Each city submitted a work plan, which included the project description, organization, operation plan, and reporting mechanisms, and the Quality Assurance (QA) plan. These work plans required more than one year to complete and obtain Regional approval. In the meantime, the projects were staffed and made operational. Community relations programs were initiated that began the process of recruiting the study participants. Coordination between the three cities was accomplished through a series of workshops, organized and convened by ECAO/RTP, approximately three per year.

This integrated assessment includes a review of the hypotheses and study designs of the individual studies (Chapter 2), a report of the methods intercomparison and quality assurance/quality control program (Chapter 3), a summary of the individual study results and conclusions reported by the three cities (Chapter 4), a description and explanation of the statistical procedures performed as part of this EPA integrated assessment and the results of these procedures (Chapter 5), and a summary of key findings and conclusions derived from this assessment (Chapter 6).

2.2 INTEGRATION OF THE THREE STUDIES

2.2.1 Study Hypotheses

To place this project in perspective, it is helpful to look at the similarities and differences among the three studies. They are similar in that their hypotheses and study designs were derived to evaluate the same general hypothesis, namely, that removing lead from soil will reduce lead exposure of young children.

The central hypothesis of the USLADP is

> *A reduction of lead in residential soil accessible to children will result in a decrease in their blood lead levels.*

Each study chose to develop a specific hypothesis that could be tested by data and observations from their own study design. The formal statement of the Boston hypothesis is

> *A significant reduction (equal to or greater than 1,000 μg/g) of lead in soil accessible to children will result in a mean decrease of at least 3 μg/dL in the blood lead levels of children living in areas with multiple possible sources of lead exposure and a high incidence of lead poisoning.*

The Baltimore hypothesis, stated in the null form, is

> *A significant reduction of lead (≥ 1,000 μg/g) in residential soil accessible to children will not result in a significant decrease (3 to 6 μg/dL) in their blood lead levels.*

The Cincinnati hypothesis, separated into two parts, is

> *(1) A reduction of lead in residential soil accessible to children will result in a decrease in their blood lead levels.*
>
> *(2) Interior dust abatement, when carried out in conjunction with exterior dust and soil abatement, would result in a greater reduction in blood lead than would be obtained with interior dust abatement alone, or exterior dust and soil abatement alone.*

Secondary hypotheses in the Cincinnati study are

> *(3) A reduction of lead in residential soil accessible to children will result in a decrease in their hand lead levels.*
>
> *(4) Interior dust abatement, when carried out in conjunction with exterior dust and soil abatement, would result in a greater reduction in hand lead than would be obtained with interior dust abatement alone, or exterior dust and soil abatement alone.*

2.2.2 General Study Design

The project objective was to measure the relationship between soil lead and blood lead. This is an indirect relationship in the sense that children most commonly do not eat soil

directly but usually ingest small amounts of dust derived, in part, from this soil. Likewise, the lead in blood reflects not only recent exposure from all environmental sources, but the remobilization of lead from bone tissue.

Each study was designed around the concept of participating families within a definable neighborhood. There were a total of twelve neighborhoods in the project, six in Cincinnati, four in Boston, and two in Baltimore. Except in Boston, these neighborhoods constituted the treatment and control groups in the study. In Boston, families in the treatment group were randomly assigned from volunteers from each of the four neighborhoods, as were families in the control group. For each treatment group, there was a preabatement, abatement, and postabatement phase. The immediate residential environment of the child was extensively evaluated prior to and after abatement, through measurements of lead in soil, dust, drinking water, and paint, and through interviews about activity patterns, eating habits, family activities, and socioeconomic status. Parallel environmental and biological measurements, as well as interviews, were taken in the control groups, but without abatement. The objective of the preabatement phase was to achieve a clear understanding of the exposure history and status (stability of the blood lead and environmental measures) prior to abatement. During the abatement phase, attention was given to preventing any possible exposure that might result from the abatement activities. During the postabatement phase, the project was designed to determine the duration of the effect of soil abatement and to detect possible recontamination.

The array of treatment groups differed considerably among the three studies. Each treatment group, however, had several features in common. All groups were taken from demographically similar neighborhoods with some prior evidence of elevated lead exposure, usually a greater than average number of public health reports of childhood lead poisoning. Each group received the same pattern of treatment: baseline phase for 3 to 18 months, intervention (except for controls), and follow-up for 12 to 24 months.

In each treatment group, even the controls, there was an attempt to minimize the impact of chipping and peeling lead-based paint. In Boston, this was done by paint stabilization of interior paint. In Baltimore, only exterior paint was stabilized. Therefore, in these two studies, the effects of soil abatement should be evaluated in the context of some intervention for lead-based paint. In Cincinnati, most of the living units were abated of lead-based paint

more than 20 years before the start of the study. In the case of those that had lead-based paint, the lead-based paint was measured but not treated prior to the study.

The Boston and Baltimore studies used a parallel intervention scheme, compared to the staggered scheme used in Cincinnati. In other words, intervention in Boston (and Baltimore) took place at the same time for all treatment groups, and the follow-up period was of the same duration. But in Cincinnati, the soil and exterior dust intervention was delayed for three neighborhoods, such that follow-up varied between 12 and 24 months. Throughout all phases of each study, the timing of the blood lead measurements was planned according to a seasonal cycle of blood lead levels that peaks in the late summer and according to an age-related pattern that peaks at 18 to 24 months.

The complex nature of this project required measurement of exposure indices, such as street dust, house dust, and hand dust, that are in the pathway between soil and blood. New sampling and analysis protocols for these measurements, not generally available in the scientific literature, were developed during the initial coordinating workshops.

The studies differ in several respects. The two pathways, (1) soil → exterior dust and (2) paint → house dust, differ slightly among the studies, as do the intervention strategies to interrupt the flow of lead along these pathways. Collectively, these differences in study design broaden the scope of the project to cover aspects of lead exposure intervention not possible through the study of a single neighborhood or even a single city.

2.2.3 Study Groups

Variations in the nature and form of intervention were included in the study designs to take advantage of the unique characteristics of the cities and their housing types. For example, soil lead concentrations are typically high in Boston, where it is also common to find elevated concentrations of lead in drinking water and in both exterior and interior paint. In the areas studied, housing is typically multi-unit with some single family units with relatively large soil cover in accompanying yards. In the Baltimore neighborhoods, nearly every house had lead-based paint, the houses were mixed single and multifamily, and the soil areas were smaller, typically less than one hundred square meters. On the other hand, houses in Cincinnati were selected because they were thought to be relatively free of interior lead-based paint that might obscure the contribution of soil lead to house dust lead. As it

happened, these neighborhoods were mostly multifamily housing with little or no soil on the residential parcel of land. The Cincinnati study design therefore focused on intervention at the neighborhood scale, where the soil in parks, play areas, and other common grounds was abated, and exterior dust on paved surfaces in the neighborhood removed.

Detailed information on study design and methods of analysis can be found in the appended individual reports for each city. Table 2-1 summarizes the study design characteristics for each of the three studies and their respective neighborhood groups. The nomenclature for these groups has been standardized for this report. With the exception of the Cincinnati control group (CIN NT), all groups received some form of intervention during the study.

For the purposes of consistency, certain descriptive terms that are used differently in the three individual study reports, are standardized here and described in the glossary of this document. One example is the use of the terms "study" and "project". In order to avoid confusion, the term "study" refers to one of the three separate community studies, and the term "project" is used in reference to the three studies collectively. Similarly, the collective term for "treatment group" or "control group" in this report is "study group".

The names that identify the individual study groups have been modified in this report to assist the reader in remembering the type of intervention performed on each group. Table 2-1 lists these names, with a brief description and the corresponding term in the report of each separate study. This nomenclature identifies location of the study and the nature of the intervention. For example, BOS SPI refers to the Boston group that received Soil, Paint, and Interior dust intervention. A hyphen is used to indicate intervention in two different rounds, as in CIN I-SE, where interior dust abatement took place about one year before soil and exterior dust abatement. The reader should become familiar with this nomenclature for the ten study groups in the project, as the data and results will be presented using these designations without further explanation. One further note: The BOS PI, BOS P, and CIN NT groups each received soil abatement at the end of the study. Because no data were reported following this intervention, the designation "-S" was not used.

Other departures here from the terminology of the respective individual study reports are conversion to a common system of units (metric where possible) and standard terms for phases, stages, or rounds of the project. The term "round" refers to a distinct period of

TABLE 2-1. TREATMENT GROUP NOMENCLATURE WITH CROSS-REFERENCE TO INDIVIDUAL REPORTS

Treatment Group Name[a]	Cross-Reference to Individual Study Report	Description of Treatment
BOSTON		
BOS SPI	Study Group	Soil and interior dust abatement, and interior paint stabilization at beginning of first year, no further treatment.
BOS PI-S	Control Group A	Interior dust abatement and interior paint stabilization at beginning of first year. Soil abatement at beginning of second year.
BOS P-S	Control Group B	Interior paint stabilization at beginning of first year. Soil abatement at beginning of second year.
BALTIMORE		
BAL SP	Study Area	Soil abatement and exterior paint stabilization at beginning of first year, no further treatment.
BAL P1[b]	Control Area	Exterior paint stabilization at beginning of first year; no further treatment.
BAL P2[b]	Study Area Not Abated	Exterior paint stabilization at beginning of first year, no further treatment because soil not above cut-off level.
CINCINNATI		
CIN SEI (P)	Area A	Soil, exterior dust, and interior dust abatement at beginning of first year, no further treatment. Includes only the Pendleton neighborhood.
CIN I-SE (B,D,F)[c]	Area B	Interior dust abatement at beginning of first year, soil and exterior dust abatement at beginning of second year, no further treatment. Includes the Back St., Dandridge, and Findlay neighborhoods.
CIN NT (G,M)	Area C	No treatment; soil and interior dust abatement following last sampling round. Includes the Glencoe and Mohawk neighborhoods.

[a]The treatment group designation indicates the location of the study (BOS = Boston, BAL = Baltimore, CIN = Cincinnati), the type of treatment (S = soil abatement, E = exterior dust abatement, I = interior dust abatement, P = loose paint stabilization, NT = no treatment).

[b]Treated as one group in the Baltimore report, analyzed separately in this report.

[c]Treated as one group for many analyses in the Cincinnati report, analyzed as individual neighborhoods in this report.

time when one or more measurements were made. Other activities, such as soil abatement, occurred between rounds. There is no consistent pattern for when abatement occurred (i.e., after Round 1, Round 3, etc.) for the different individual cities.

The numbers of participating children, families, and properties appear in Table 2-2. Because of attrition and recruitment in Baltimore and Cincinnati, these numbers do not accurately represent the number of participants present for the duration of the study. In this report, subsets of these participants were statistically analyzed for specific purposes and to meet specific statistical requirements, and these subsets may not be the same subsets used by the individual study teams in their statistical analysis described in their respective individual city reports.

2.2.4 Project Activity Schedule

The project activity schedule, shown in Figure 2-1, illustrates the major intervention and measurement activities of the individual studies and the sequence and duration of these activities. The frequency and timing of sampling relative to abatement and seasonal cycles are important issues in the study design. These time lines are the actual occurrence of these events and they differ somewhat from the planned schedule. The original design focused on sampling blood lead during the late summer, as it was known that the seasonal cycle for blood lead reaches a peak during this period.

2.2.5 Environmental and Biological Measurements of Exposure

Figure 2-2 illustrates the generalized concept of the pathways and sources of human exposure to lead, showing the routes of lead from the several sources in the human environment to the four compartments (inhaled air, dust, food, drinking water) immediately proximal to the individual child. One of these proximal sources, dust, is the primary route of concern in this project. Figure 2-3 expands this dust route to show both the complexity of the many routes of dust exposure for the typical child and the mobility of dust lead along these routes. Both of these concepts were poorly understood in the late 1980's. The intervention strategies used in this project were designed to interrupt the movement of lead along one or more of these pathways.

TABLE 2-2. NUMBER OF PROJECT PARTICIPANTS BY TREATMENT GROUP AND ROUND[a]

BOSTON	Treatment Group	R1 (PRE)	R2 (POST 1)	R3 (POST 2)	R4 (Phase 2)
Middate of round		10/17/89	4/9/90	9/12/90	7/20/91
Children[b]	BOS SPI	52	52	52	33
	BOS PI-S	51	48	49	33
	BOS P-S	47	46	46	26
		150	146	147	92
Families[c]	BOS SPI	43	43	43	28
	BOS PI-S	43	40	41	27
	BOS P-S	39	38	38	22
		125	121	122	77
Properties[d]	BOS SPI	34	34	34	24
	BOS PI-S	36	33	34	24
	BOS P-S	30	29	29	19
		100	96	97	87

BALTIMORE	Treatment Group	R1	R2	R3	R4	R5	R6
Middate of round		10/25/88	4/1/89	2/17/90	1/27/91	6/7/91	9/3/91
Children[b]	BAL SP	88	85	110	103	99	95
	BAL P1	73	73	80	79	80	79
	BAL P2	7	7	8	8	7	8
		168	165	198	190	186	182
Families[b]	BAL SP	63	60	72	68	65	64
	BAL P1	50	50	52	51	51	51
	BAL P2	6	6	7	7	6	7
		119	116	131	126	122	122
Properties[b]	BAL SP	55	53	61	59	53	57
	BAL P1	45	45	47	46	46	46
	BAL P2	6	6	7	7	6	7
		106	104	115	112	105	110

CINCINNATI	Treatment Group	R1 (P01)	R3 (P03)	R4 (P05)	R6 (P07)	R7 (P09)
Middate of round		7/6/89	11/14/89	7/1/90	11/17/90	6/16/91
Children[b]	CIN SEI (P)	54	52	46[f]	37	31
	CIN I-SE (B,D,F)	86[e]	81[e]	92[f]	87	77
	CIN NT (G,M)	61	52	81[f]	74	61
		201	185	219	198	169
Families[c]	CIN SEI (P)	31	30	31	31	30
	CIN I-SE (B,D,F)	58[e]	56[e]	56	74	60
	CIN NT (G,M)	40	37	35	63	52
		129	123	122	168	142
Parcels[d]	CIN SEI (P)	55	39	39	40	40
	CIN I-SE (B,D,F)	74[e]	121[e]	121	119	121
	CIN NT (G,M)	86	85	85	84	84
		215	245	245	243	245

[a] Round designations (R1, R2, etc.) are not the same as used in the Boston and Cincinnati study reports. Their round designations are shown in parentheses. Some rounds are omitted from this table because blood lead data were not collected. Intervention, shown by the dashed lines, occurred between R1 and R2 in the first year and R3 and R4 in the second year in Boston, R3 and R4 in Baltimore, R1 and R3 in the first year of the Cincinnati study, and R4 and R6 in the second year. Middates are the mean blood sampling dates.

[b] Based on number of children sampled for blood.

[c] Based on number of households sampled for dust.

[d] Based on number of soil areas sampled.

[e] Dandridge was added to the Cincinnati study after the soil sampling for R1, but before the completion of all other R1 sampling. Thus, the number of Dandridge children and families are included in R1 for CIN I-SE, but the number of parcels are not included until R3.

[f] These numbers reflect additional children recruited from participating families in July, 1990. The Cincinnati report does not include these children.

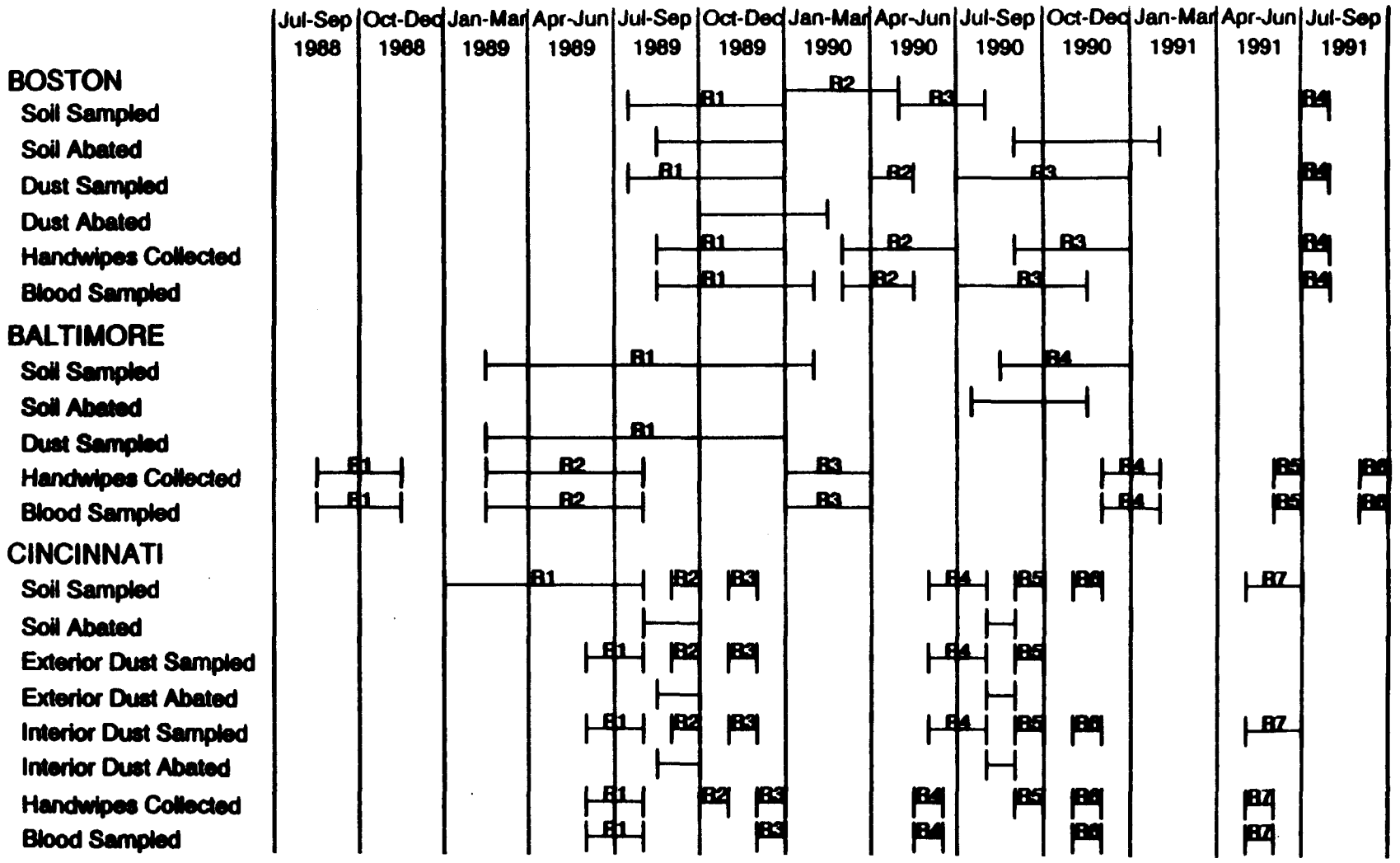

Figure 2-1. Project activity schedule showing the round designations and time periods for sampling and interviewing, and the time periods for soil abatement. Paint stabilization in Boston and Baltimore was performed during the soil abatement period prior to any other intervention. Abatement in Cincinnati that was performed after the final sampling round (as a sampling round (as a courtesy to participants) is not shown in this figure.

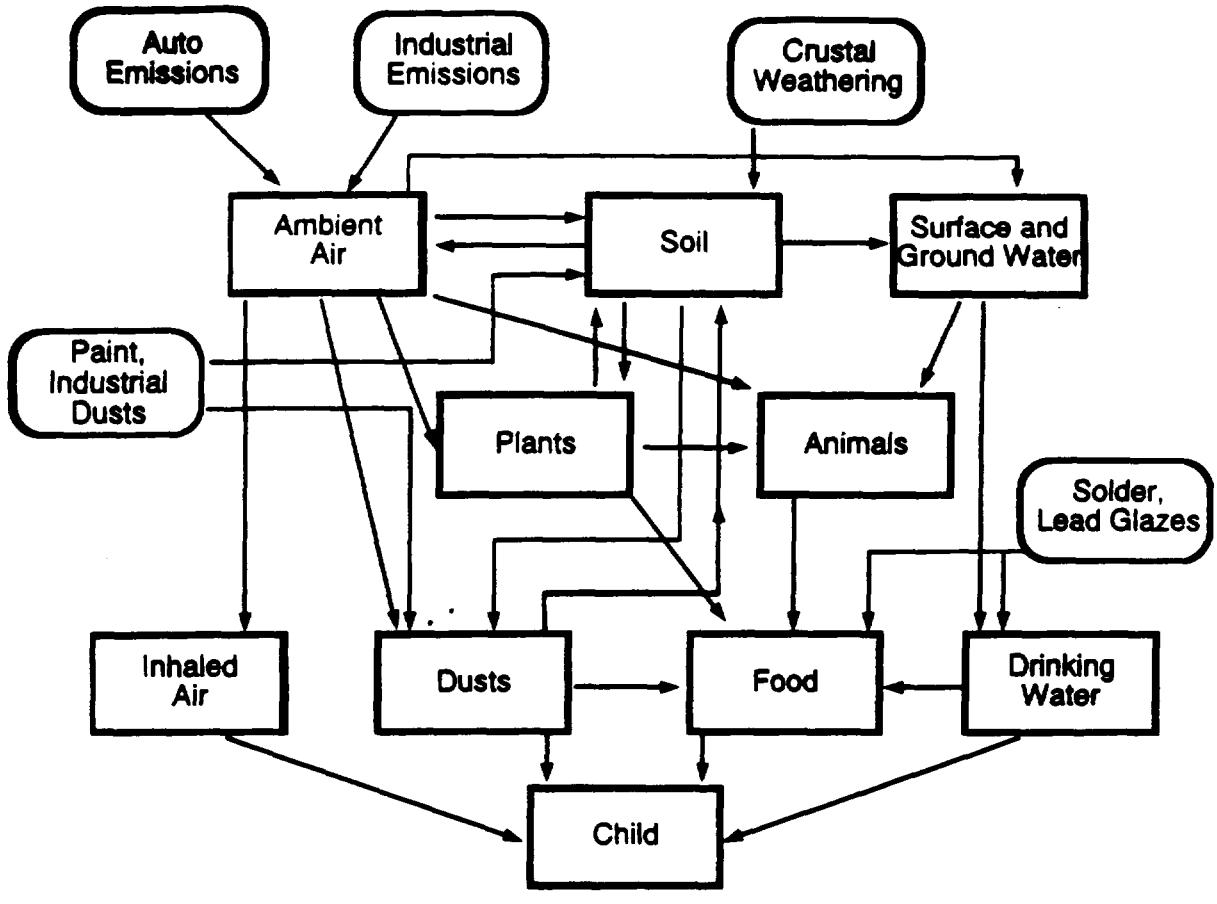

Figure 2-2. Generalized concept of the sources and pathways of lead exposure in humans.

Exposure is the amount of a substance that comes into contact with an absorbing surface over a specific period of time. In the case of lead, the absorbing surface can be the gastrointestinal tract or the lungs. Exposure is measured in micrograms of lead per day. Thus, an exposure of 10 μg/day represents a total ingestion and inhalation of 10 micrograms of lead from all sources; a fraction of this 10 micrograms would be absorbed into the body.

2.2.5.1 Blood Lead

In this project, blood lead was used as an indicator of exposure, and reductions in blood lead concentrations were expected as a result of any combination of the interventions described above. The units for blood are micrograms of lead per deciliter of blood (μg/dL) and they are not compatible with the normal units of exposure, micrograms of lead per day.

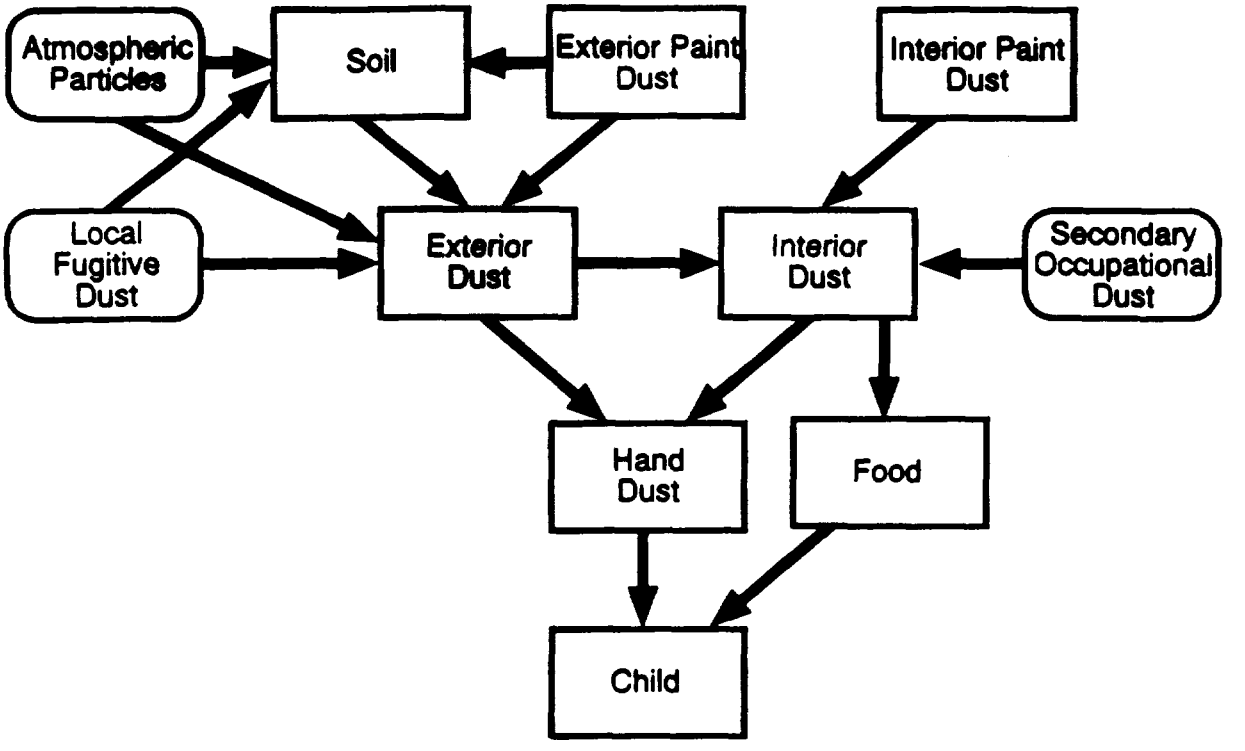

Figure 2-3. Typical pathways of childhood exposure to lead in dust showing both the complexity of the routes of exposure and the mobility of dust lead along these routes.

The fraction of ingested lead that is actually absorbed in the gastrointestinal tract depends in part on the bioavailability of the particular form of lead. The amount of absorbed lead that reaches specific body tissues depends on the biokinetics of lead in the human body. Blood tissue is in dynamic equilibrium with all other body tissues, including bone tissue, where the lead is stored for longer periods of time.

The relationship between blood lead concentration and the onset of health effects of lead, depends largely on the distribution of lead to the target tissues, including the red blood cells themselves. Blood lead, then, is a convenient indicator of both exposure and potential health risk to the child. This situation becomes important when measuring the rate at which blood lead concentrations might decline following abatement. For a child with lead stored in bone tissue following a long history of high lead exposure, the decline in blood lead might be expected to be slower than for a child with low previous exposure. Even if lead-burdened children were moved to an environment completely free of lead exposure, a significant

amount of lead would still be present in the child's blood due to the slow release of lead from the large amounts stored in bone and other body tissues.

Autopsy data show that as much as 60 to 70% of the lead in a child's body is stored in the skeletal system, especially in the hard (or cortical) part of long bones such as the femur and the tibia (Barry, 1981). In adults this percentage is even larger, 90 or 95%. Lead is retained in cortical bone for many years, and even though bone remodeling in young children is very rapid, these large body burdens contained in the bone constitute a significant internal source of lead exposure for several years after exposure has stopped. The long-term stability of blood lead levels in a stationary exposure environment has been noted by a number of authors (David et al., 1982; Rabinowitz, 1987).

Persistence of elevated blood lead after abatement has both biological and environmental components. The biological component is the resorption of skeletal lead. In adults, recent stable lead isotope studies (Smith et al. 1995, 1996; Gulson et al., 1995) suggest that 30 to 65% of the circulating lead in adults is due to skeletal lead, which is consistent with other estimates. Similar studies have not been reported for children. Although a somewhat lower percentage may be appropriate for children rather than adults, it is clear that even in children a substantial fraction of blood lead has a skeletal origin.

The environmental component of persistence is the child's remaining exposure to other nonremediated lead media, such as lead in diet, drinking water, or air. This is illustrated in Figure 2-4, which shows a blood lead profile (for an individual, or possibly as a population mean) before and after a hypothetical lead abatement. The steady-state blood lead concentrations are shown as flat curves, although in reality there may be substantial age-dependent changes during the course of abatement even when environmental lead concentrations remain constant. Assuming that environmental concentrations remain constant after abatement, the child's blood lead would eventually reach a new steady-state concentration at a much lower level. At any given time after abatement, the child's blood lead is a mixture of three components, denoted "A", "B", and "C" in Figure 2-4. Component A shows the relatively rapid decrease in blood lead from elimination of preabatement lead deposits in blood and soft tissues. Component B shows the contribution of preabatement skeletal lead to post-abatement blood lead, which is much slower because the large skeletal burden in cortical bone is eliminated on a time scale of several years. Almost

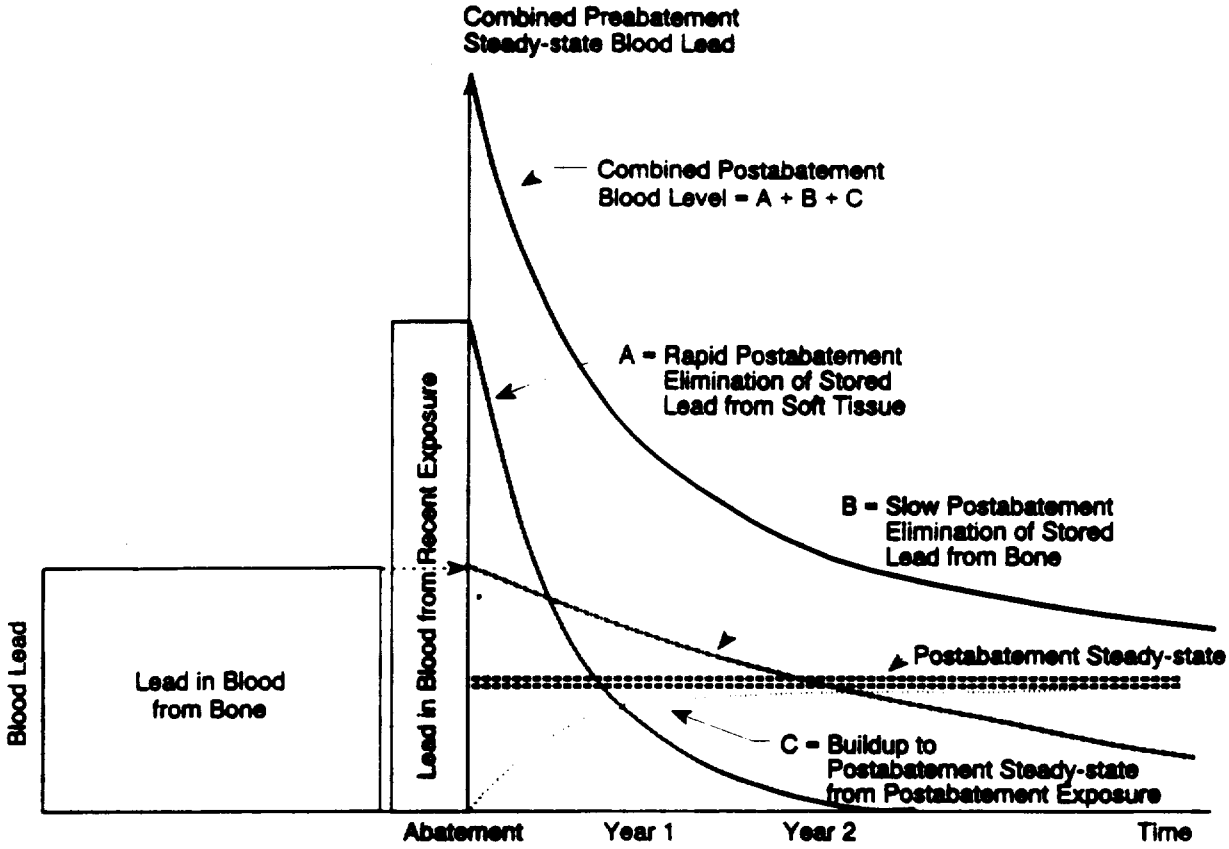

Figure 2-4. Hypothetical representation of the expected decrease in blood lead (solid curved line) following abatement. This rate of decrease is less than might be expected from exposure reduction alone. This is because blood also contains lead recently released from storage in bone and soft tissue.

all of the stored lead may eventually be eliminated. However, the contribution of preabatement deposits of lead now stored as an internal source of exposure may be quantitatively significant compared to remaining postremediation environmental exposure media.

The combination of persistent internal exposure and persistent baseline external exposure amounts to a post-abatement blood lead contribution of about 50 or 60% of the preabatement blood lead starting value at 8 to 12 months after abatement. This suggests that under optimum conditions any environmental abatement or intervention can likely only achieve a 40 to 50% reduction in child blood lead concentrations within a year after abatement (see Figure 2-4).

Several authors have reported differences in persistency of elevated blood lead concentrations between smelter and non-smelter communities (Angle et al., 1984; Gallacher et al., 1984a,b; Roels et al., 1980, 1976; Angle and McIntire, 1979; Yankel et al., 1977). In general, blood lead concentrations in non-smelter children tend to decrease at ages beyond four years, whereas smelter children usually retain childhood pattern of elevated blood lead into their teens. This difference has been attributed by Mushak (1993) to the nature and disposal of smelter emissions. In general, the hypothesis is that urban children older than four years should show lower blood lead concentrations than they did at age 2 to 3 years. This hypothesis can be tested with the data from the present studies, but the hypothesis that smelter children differ from urban children cannot.

2.2.5.2 Hand Lead

Because blood lead reflects exposure to lead from all environmental sources, a second exposure indicator, hand lead, was used to focus directly on the immediate pathway of dust to the child. The units of measure are micrograms of lead per pair of hands, and like blood lead, this measure does not reflect the rate at which lead moves into the body in units of micrograms of lead per day. Instead, this hand dust is a measure of lead loading on the hand. It is a measure of the "dirtiness" of the hand in the same sense that dust loading is a measure of the dirtiness of the floor. Hand dust loading could possibly be converted to micrograms of lead per day if there were a measure of the area of the hand mouthed by the child, the frequency of hand to mouth activity, and the frequency of hand washing during each day.

2.2.5.3 House Dust

House dust is a mixture of lead from many sources, including soil, street dust, interior paint, and occupational dusts carried home by family workers. The units of measurement are μg Pb/g (lead concentration), μg Pb/m^2 (lead loading), and mg dust/m^2 (dust loading). When expressed as micrograms of lead per gram, the measurement can be converted to an exposure measurement by assuming a specific amount of dust ingested per day, usually about 100 mg/day for preschool children. Exposure to household dust then becomes micrograms per day:

$$Pb\ Concentration \times Ingestion = Exposure$$

$$\frac{\mu g Pb}{g\,dust} \times \frac{g\,dust}{day} = \frac{\mu g Pb}{day}. \qquad (2\text{-}1)$$

In a similar manner, exposure to food, drinking water, and inhaled air can be expressed as μg/day, and in 1990 these three sources normally accounted for about 5, 1, and 0.1 μg Pb/day respectively (U.S. Environmental Protection Agency, 1994). If the lead concentration in household dust is 200 μg/g and dust ingestion is 0.1 g/day, the exposure is 20 μg/day or much more than the other sources combined.

By a different calculation, childhood lead exposure may be expressed as a function of dust lead loading. In this case, the ingestion parameter is in units of m^2/day:

$$Pb\ Loading \times Ingestion = Exposure$$

$$\frac{\mu g Pb}{m^2} \times \frac{m^2}{day} = \frac{\mu g}{day}. \qquad (2\text{-}2)$$

The ingestion parameter estimates the effective contact area for the child's hands (assuming all dust is ingested by hand-to-mouth activity). Literature reports of childhood lead exposure based on contact area are not known.

2.2.6 Intervention Strategies

Intervention is defined here as the interruption of the flow of lead along an exposure pathway. Soil abatement is one form of intervention. If done correctly, this abatement should establish an effective and persistent barrier to the movement of lead through the child's exposure pathways. Other forms of intervention used in this project were exterior dust abatement, interior dust abatement, and paint stabilization. Because dust is a very mobile constituent of the human environment, exterior and interior dust abatement would not be expected to form a permanent barrier to lead unless other sources of lead, such as soil, were also abated. Likewise, the form of paint stabilization used in Boston and Baltimore, where chipping and peeling paint was removed and the walls repainted, was not intended to be permanent lead-based paint abatement.

The strategy for soil abatement was to remove all soil with concentrations above a specific level (500 μg/g for Baltimore and Cincinnati, 1,000 μg/g for Boston), and replace this soil with clean soil in the range of 25 to 100 μg/g lead concentration. This method (excavation, removal, and replacement) was used in all three studies. In some cases, repair and maintenance of ground cover was used where the soil concentrations did not warrant excavation, removal, and replacement. To further interrupt the flow of lead along the exposure pathways, entire neighborhoods in Cincinnati were cleaned of exterior dust using street cleaning vacuum equipment and hand tools.

Interior house dust is believed to be a major direct lead exposure pathway for children. Because household dust typically contains a mixture of lead from several sources (e.g., soil, interior/exterior paint, air, etc.), abating house dust temporarily separates such sources from the child's environment. Their recontamination of house dust and consequent impact on the child's lead exposure can be evaluated by comprehensive measurements of the household dust that include changes in lead concentration, lead loading, and dust loading. Understanding the expected impact of abatement on these three parameters is critical to interpreting the observed changes in blood lead concentrations. Following dust abatement, there should be an immediate decrease in the dust loading, with no change in the lead concentration for those groups that did not receive soil, exterior dust, or paint intervention. The rate at which this dust loading returns to preabatement levels reflects the rate of movement of dust from other sources into the home, the frequency of cleaning, and the "cleanability" of the home. (Many inner city homes have surfaces that are cracked, pitted, or in disrepair and are difficult to clean effectively.)

The effectiveness of both paint stabilization and soil and dust abatement can be observed by changes in the lead concentrations of house dust. In the presence of lead-based paint, the concentration of lead in house dust is expected to be greater than 1,500 to 2,000 μg/g, whereas without the influence of lead-based paint, the house dust is expected to be comparable to external dust and soil (U.S. Environmental Protection Agency, 1986).

House dust is a mixture of dusts from many sources within and outside the home. In the absence of lead-based paint inside the home, it would seem reasonable to assume that most of the lead in household dust comes from soil and other sources external to the home.

Therefore, to enhance the impact of soil abatement, interior dust abatement was carried out for some treatment groups in Boston and Cincinnati.

Many of the Boston and Baltimore households selected for the project had chipping and peeling paint, both interior and exterior. In order to reduce the impact of lead-based paint, the walls and other surfaces were scraped and smoothed, then repainted. It is important to note that no attempt was made to remove all lead-based paint, nor to isolate intact paint from the child. Paint stabilization was used on interior surfaces in Boston and on exterior surfaces in Baltimore. Paint stabilization was not used in Cincinnati because most of the lead-based paint was believed to have been removed from most of these homes in the early 1970s.

2.3 EXTERNAL FACTORS THAT COULD INFLUENCE PROJECT RESULTS AND INTERPRETATION

The Scientific Coordinating Panel recognized that several extraneous factors might influence the outcome of the project and that these factors were generally beyond the control of the investigators. Among these are seasonal cycles and time trends of childhood blood lead concentrations, unexplained or unexpected sources of lead in the children's homes or neighborhoods, changes in public perception and avoidance of lead exposure hazards, and movement of lead in soil either down the soil column or laterally with surface runoff or as fugitive dust.

2.3.1 Cycles and Trends in Environmental Lead Concentrations

Figure 2-5 illustrates a pattern of childhood blood lead concentrations for Chicago during the 1970s, showing a seasonal cycle and a downward trend throughout the decade. The National Health Assessment and Nutrition Examination Survey II (NHANES II) data for the entire country and all age groups reported a similar seasonal cycle and downward trend during the last half of that decade (Annest et al., 1983). (Seasonal patterns from the NHANES III data of 1988 through 1991 are not yet available.)

Investigators have known about this seasonal pattern for some time (Figure 2-6). Most epidemiological studies are planned so that measurements can be taken at the peak of this cycle, generally during the late summer. Studies of large numbers of children show a

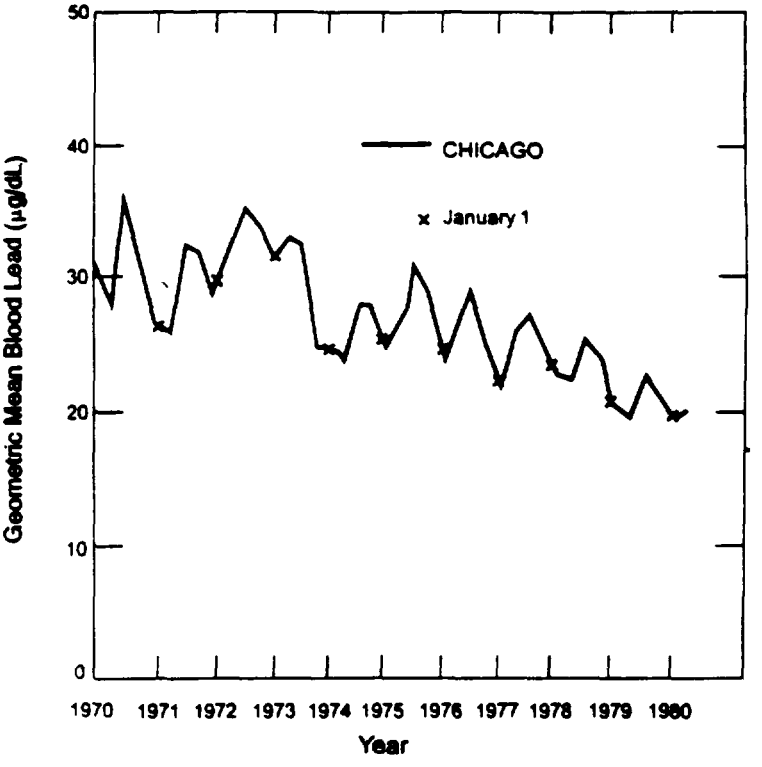

Figure 2-5. Literature values for seasonal patterns for childhood blood lead (age 25 to 36 mo) in Chicago. These data generally show an annual peak blood lead during late summer.

Source: U.S. Environmental Protection Agency (1986).

sinusoidal pattern, even when the measurements do not include sequential measurements for the same child. During the development of the study designs, it was apparent that understanding of the seasonal cycles and temporal trends in blood lead would play an important part in the interpretation of data collected over several years.

There is a question as to whether the seasonal cycle for blood lead concentrations is caused by fluctuations in exposure or by physiological processes that regulate the biokinetic distribution of lead within the body. Some investigators have attributed fluctuations in blood lead concentrations to changing environmental lead concentrations or changing activity patterns (U.S. Environmental Protection Agency, 1995). During the late summer months, the child may eat food or dust with high lead concentrations or ingest more dust during outdoor play. This project was designed to measure changes in lead concentrations in soil and dust, but not changes in activity patterns.

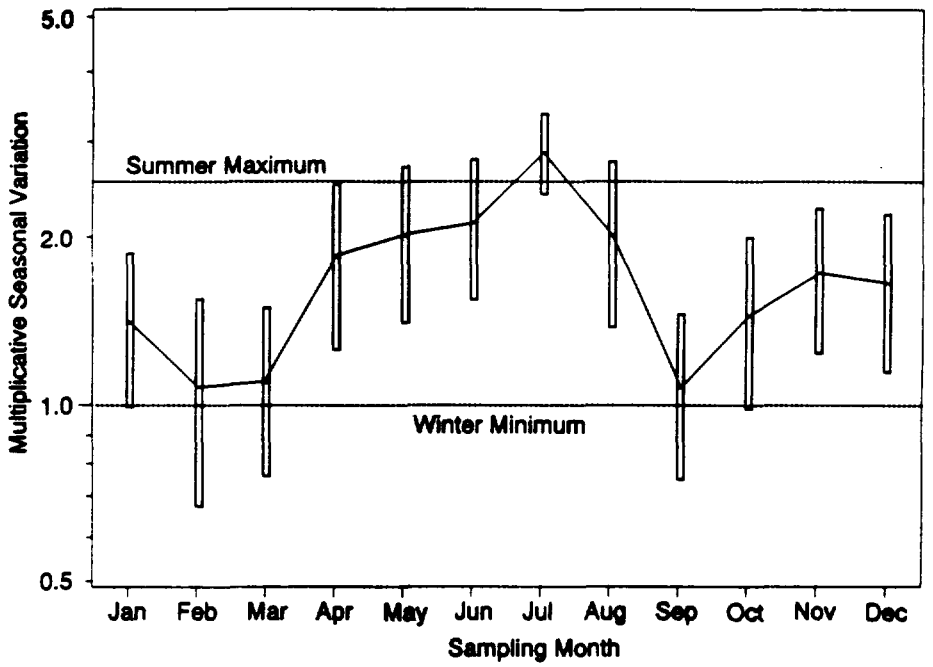

Figure 2-6. Estimated seasonal variation based on residual blood-lead levels in Boston children after controlling for age and date of birth effects. (Bars represent 95% confidence bounds for blood-lead residuals.)

Source: United States Environmental Protection Agency (1995).

Although this project was designed to maximize the measurements of blood lead during the late summer for each of the three studies, measurements were made during other times of the year in order to observe changes immediately after abatement. For most statistical analyses in this report, comparisons were made from measurements taken approximately twelve months apart in order to minimize the impact of the seasonal effect. A more detailed description of this treatment appears in Chapter 5.

Two other patterns, long-term time trends and early childhood patterns dependent on age, are applicable to this project. Little is known about age related patterns, but one study in Cincinnati, prior to the project, showed a pattern of blood lead changes during early childhood growth patterns (Figure 2-7).

Long-term downward trends were documented for child blood lead concentrations during the 1970s and 1980s and have been attributed to decreasing concentrations of lead in

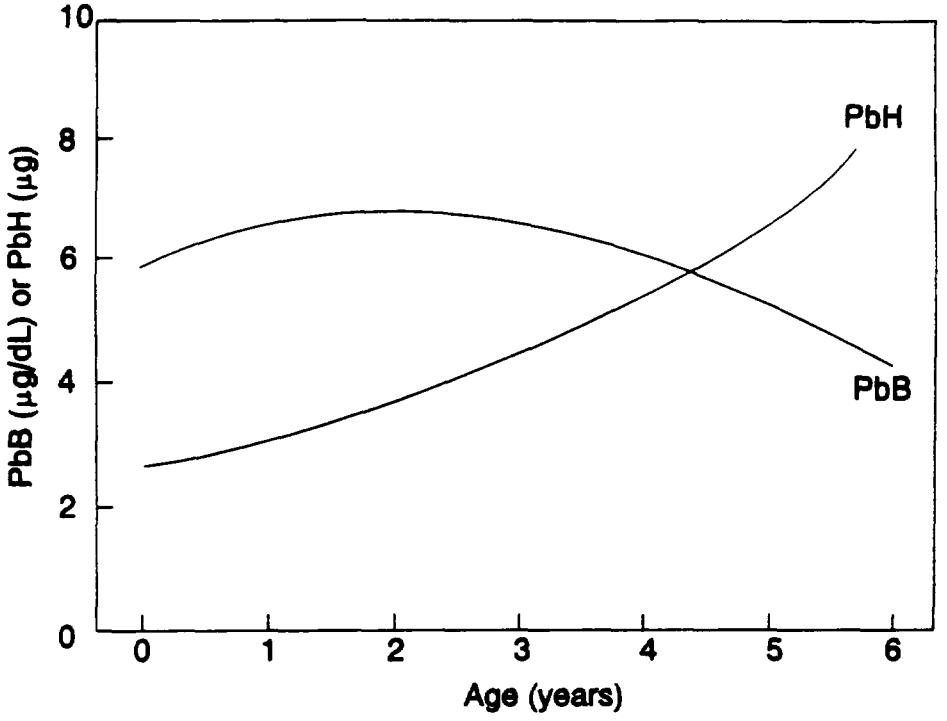

Figure 2-7. Predicted differences in blood lead (PbB) and hand lead (PbH) during early childhood, based on empirical data. The peak for blood lead at age 2 may be due to activity patterns related to dust ingestion for toddlers and young children. The steady increase in hand lead could be due to the increase in hand size as well as activity patterns favoring play outside the home.

Source: Bornschein et al. (1985).

food and air. The QA/QC measures reported in detail in Chapter 4 rule out the possibility of this trend being caused by a measurement artifact such as analytical drift.

2.3.2 Unexplained and Unexpected Sources of Lead

Occasionally, measurements of environmental lead are higher than expected and difficult to explain. Atmospheric deposition can be a reasonable explanation, because this route can change much more abruptly than soil, dust, food or drinking water. This section discusses the possibility that the observed fluctuation in street dust and house dust can be attributed to changes in air concentration alone. Because this project began after the national phasedown of lead in gasoline, the air concentrations of lead in these cities had decreased to

about 0.1 $\mu g/m^3$ by the start the project.[2] The following is a theoretical calculation of the amount of lead that could be transferred to soil or dust at this concentration and from this source alone.

Atmospheric deposition during the project was assumed to be typical for air concentrations that averaged 0.1 $\mu g/m^3$ (1.0×10^{-7} $\mu g/cm^3$). At a deposition rate of 0.2 cm/s, this would accumulate 0.6 $\mu g/cm^2 \cdot$year at the soil surface. Assuming that this lead would be retained in the upper 1 cm of soil surface (therefore 1 cm^2 of soil surface equals 1 cm^3 of soil), then the annual increment would be 0.6 $\mu g/cm^3$. Because 1 cm^3 of soil weighs about 2 g, the annual incremental increase in lead concentration would be 0.3 μg Pb/g soil, an insignificant annual contribution for soils that average several hundred micrograms per gram. The calculation for annual deposition to a surface is

$$1 \times 10^{-7} \frac{\mu g\ Pb}{cm^3} \times 0.2 \frac{cm}{s} \times 3.15 \times 10^7 \frac{s}{year} = 0.6 \frac{\mu g\ Pb}{cm^2\ year}. \qquad (2\text{-}3)$$

For the accumulation of dust on hard surfaces, however, the same calculation indicates a potentially greater influence of atmospheric lead. Converting to units of lead loading, the 0.6 $\mu g/cm^2 \cdot$year becomes 6,000 $\mu g/m^2 \cdot$year, or 16 $\mu g/m^2 \cdot$day. Therefore, 0.1 $\mu g/m^3$ in air concentration could account for a change of 16 μg Pb/m^2 per day in the dust lead loading to a surface. An accumulation of 160 $\mu g/m^2$ over 10 days is in the range of the observed changes in surface dust loading in this project.

2.3.3 Movement of Lead in Soil and Dust

There are several reasons why localized soil lead fluctuations might occur. Changes in soil lead concentration independent of intervention that might increase lead concentration are: atmospheric deposition (relatively minor as discussed above), exterior paint chipping and chalking, and human activity such as household waste dumping (motor oil, etc). Soil lead concentrations might decrease if lead leaches downward into the lower soil horizon, or if surface soil shifts by dust reentrainment. The downward leaching of lead through the soil

[2] The 1989 maximum quarterly average air lead concentration for the metropolitan statistical areas of Boston, Baltimore, and Cincinnati were 0.08, 0.11, and 0.11 $\mu g/m^3$, respectively (U.S. Environmental Protection Agency, 1991a).

profile mass occurs at a very slow rate, approximately a few millimeters per decade (Grant et al., 1990). The reentrainment of dust at the soil surface is usually in equilibrium with the local environment, such that inputs would equal outputs by this pathway. This would not be the case if there is flaking or peeling lead-based paint within the neighborhood or an industrial source of fugitive dust in the vicinity of the neighborhood. A limited effort was made to monitor and control the impact of lead-based paint on soil concentrations.
In Baltimore, buildings with exterior lead-based paint were stabilized by removal of the chipping and peeling paint, done in a manner to avoid contaminating the soil. In Boston, homes were selected with less then 30% exterior chipping and peeling paint, by area.
In Cincinnati, neighborhoods with mostly rehabilitated houses were selected. There were no attempts in any of the studies to control the introduction of lead to the soil by human activity such as household waste dumping.

Lead in household dust is a mixture of dust brought into the house from outside and dust generated from within the home. Studies have shown that as much as 85% of the mass of dust comes from outside the home and much of this is apparently brought in on the feet of children and pets (Roberts et al., 1991). Household dust lead concentrations are usually similar to the soil concentration in the immediate vicinity of the house, unless there are internal sources of lead, such as lead-based paint. Thus, changes in soil concentrations are likely to be reflected by changes in household dust concentrations within a few days and probably reach equilibrium within a few months, depending on the relative contribution from soil and other sources, the frequency and efficiency of house cleaning, and the cleanability of the house.

2.3.4 Factors That Limit Interpretation of the Project Results

In the following chapters, this report discusses several issues that identify possible limitations of the studies. This detailed assessment: (1) examines measurement methods used and related QA/QC data to ascertain that adequate measures were taken to produce data of good quality that can be compared across the three studies; (2) examines the study designs to determine if the individual study groups are comparable within each study and if comparisons are possible across the three studies; and (3) performs rigorous statistical analyses that

attempt to quantify differences between study groups and identify specific exposure factors that may be responsible for the differences.

With respect to the QA/QC data, it should be noted that there are no estimates of sampling reproducibility for any of the environmental or biological measurements. This would have required collecting duplicate samples for a specified percentage of the samples. In retrospect, the following observations are worth noting:

1. Duplicate soil samples would not have been informative unless the entire soil parcel was sampled in duplicate. In this report, the reproducible number is the arithmetic mean of all soil samples from the parcel;

2. Duplicate sampling of house dust would have identified reproducibility of lead concentration, but probably not lead loading, which changes on a daily basis. Duplicate sampling of house dust may also have impacted the child's environment if a substantial amount of the targeted play areas were sampled.

Nevertheless, this report recognizes the limitations of statistical analysis due to the absence of an estimate of sampling error.

There are several exposure-related factors other than those measured by environmental sampling that must be taken into account during the statistical analyses. Among these are seasonal patterns in weather (especially rainfall as it affects dust loading and mobility), activity patterns (which affect indoor/outdoor play patterns), and possible physiological growth cycles (which affect remobilization of lead from bone tissue). Age of the child may also impact exposure by differences in activity patterns, body size, and parental supervision. For the most part, this report is only able to assume that all groups within a study were impacted equally by these and other confounding factors during the study.

3. METHODS INTERCOMPARISON AND QUALITY ASSURANCE/QUALITY CONTROL

Specific details on measurement methodology employed in each study can be found in the appended individual city reports. This chapter describes the initial evaluation of several methods for soil, dust, hand wipe, and blood sampling and analysis that were considered by the Scientific Coordinating Committee, and the basis for selection of these methods by the participating research teams.

Soil sampling procedures were defined based on agreement that five 2-cm soil cores would be taken according to a prescribed pattern about a randomly selected point, and that a prescribed number of these points would be selected based on the size and shape of the plot of soil. These procedures are described in the individual reports, and no further assessment is made here of the representativeness of this sampling procedure.

Interior dust sampling methods were determined based on the desirability of obtaining dust loading information. This required that a dry sample be taken (as opposed to a wet wipe) in order to determine the mass of dust collected as a function of area (dust load). Although the sampling devices differed, the basic protocol called for a vacuum pump that collected the dust sample on a filter pad at a prescribed flow rate and using a prescribed pattern of moving the pump nozzle over the sample area. No further attempt was made to calibrate the collection devices between the individual studies.

Hand wipe samples were taken according to procedures developed by the Cincinnati group in previous studies. Field blanks and lot blanks were determined by each group. There were some differences in the timing of the hand wipe sample (home visit versus clinic visit) as reported by the individual study teams.

Blood samples were taken according to methods prescribed by CDC in their blood lead certification program. The analysis of blood for health indicators (FEP, TIBC, etc.) other than lead differed among the three groups. Only the blood lead concentration data were used in this integrated assessment.

The procedures and results of interlaboratory comparisons of analytical methodology and the results of the QA/QC plan for the individual studies are described in the following

sections. These procedures and their results were reviewed and evaluated throughout the project at the scheduled workshops and during monthly teleconference calls.

The research team for each study prepared a sampling and analysis plan that included rigorous QA/QC objectives. These plans included protocols that: defined sampling schemes designed to characterize the expected exposure to soil for children; described how to collect, transfer, and store samples without contamination; and described how to analyze samples with the maximum degree of accuracy and precision. Sampling protocols for soil, handwipes, and blood lead were nearly identical. Dust sampling protocols differed with regard to the vacuum device used, location of sample within the residence, and procedures for pooling samples prior to analyses. These differences may, in retrospect, have affected the comparability of both the dust load and dust lead concentration data. During the course of the project, several intercalibration exercises were performed to ensure that the analytical results for measurements of soil, dust, handwipes, and blood would be accurate and that the data would be as comparable as possible.

3.1 INTERCOMPARISON OF LABORATORY METHODS FOR SOIL AND DUST MEASUREMENTS

The objective of the laboratory intercomparison and QA/QC program was to ensure that the three studies could achieve high-quality analyses of soil and dust samples, and that each of the three laboratories would be expected to get similar results when analyzing the same soil sample. The participating cities recognized the need for standardizing the sampling and analytical protocols so that data from each study could be compared. This standardization was accomplished for soil and dust by measuring the analytical difference between each of the three labs. Common standards were prepared and a program for assuring data quality was put into place. A three step program was agreed to that involved: (1) a round robin calibration exercise of soil samples to measure differences between laboratories and differences between analytical methods and instrumentation; (2) a double blind audit system for soil and dust to monitor the performance of each laboratory during the project; and (3) a second round robin calibration exercise to determine the arithmetic correction factor that would normalize dust and soil data to a common project basis. This program ensured

that analyses performed by each of the three participating laboratories would be internally accurate and externally consistent with similar analyses by other research laboratories.

Intercalibration Exercise I was conducted prior to the beginning of each study using soil and dust samples collected from representative neighborhoods in each city. Intercalibration Exercise II was conducted near the end of the sampling phase of the project using aliquots of soil and dust samples collected at the beginning of the sampling phase, some of which were used for QA/QC monitoring during the project. In each calibration exercise, two additional laboratories were invited to participate in order to determine some measure of comparability with other studies reported in the scientific literature. All laboratories reported their results independently. In the time period between these two calibration exercises, the effectiveness of the individual QA/QC programs was also monitored by inserting double blind audit samples into the sample stream of each study to measure the consistency of analytical precision throughout the study and to monitor any analytical drift.

3.1.1 Round Robin Intercalibration Exercise I

At the beginning of this project, the methods proposed by each study for soil and dust analysis were reviewed by the Scientific Coordinating Panel. The preferred method, hot nitric acid digestion followed by atomic absorption spectroscopy (AAS), was time consuming and expensive. The number of samples was expected to exceed 75,000 per study, so more rapid and less expensive methods were evaluated. Laboratory scale X-ray fluorescence (XRF) spectroscopy and inductively coupled plasma (ICP) emission spectroscopy were proposed, and a cold nitric acid extraction method for AAS was also considered.

In May 1988, prior to the beginning of each study, each of the three laboratories collected ten soil samples from areas similar to those that would be included in their study. One of the samples from Cincinnati was a street dust sample of very high lead concentration. The other 29 samples were selected from soils with lead concentrations expected to range from 250 to 8,000 μg/g. The samples were dried and sieved according to the study protocols. Approximately 200 g of each sample were sent to the other two laboratories and to an outside laboratory at Georgia Tech Research Institute (GTRI). Table 3-1 shows the instrumentation and method of analysis used by each laboratory. In making these analyses, each laboratory used its own internal standards for instrumental calibration and shared a

TABLE 3-1. WET CHEMISTRY AND INSTRUMENTAL METHODS USED FOR THE FIRST INTERCALIBRATION STUDY

	Participating Laboratories				
Method[a]	Boston	Baltimore	Cincinnati	GTRI[b]	USDA[c]
Hot HNO_3/AAS		X	X		
Cold HNO_3/AAS			X		X
Hot HNO_3/ICP		X			
XRF	X			X	

[a]HNO_3 = Nitric acid; AAS = Atomic absorption spectroscopy; ICP = Inductively coupled plasma emission spectroscopy; XRF = X-ray fluorescence.
[b]GTRI = Georgia Tech Research Institute.
[c]USDA = U.S. Department of Agriculture.

common set of five standards provided by Dr. Rufus Chaney at the U.S. Department of Agriculture. The intercalibration exercise successfully established a baseline for cross study comparison of soil and dust results.

In summary, the test conditions were that each laboratory would be provided with instructions for preparing the samples (drying, sieving, and chemical extraction) but would use their own internal standards and instrumental settings. They would have access to a set of external standards (from U.S. Department of Agriculture) with known values from which they could make corrections if necessary.

Each of the three study laboratories sent aliquots of 10 samples to the other two participating laboratories and to two external laboratories. The samples were subdivided by sieving during preparation to a "total" and "fine" fraction. Thus there were 30 samples, each with two size fractions analyzed by each of five laboratories using either one or two analytical methods, as indicated in Table 3-1. The results of the analyses appear in Table 3-2.

The cold nitric acid extraction method was found to be essentially equivalent to the hot nitric acid extraction method for soils with lead concentrations up to 8,000 $\mu g/g$ (Figure 3-1) for the samples analyzed in this study. The AAS method used by Cincinnati and Baltimore was also equivalent (Figure 3-2), showing a high degree of comparability between these two laboratories under these test conditions.

TABLE 3-2. ANALYTICAL RESULTS OF THE FIRST INTERCALIBRATION STUDY: LEAD CONCENTRATION (μg/g) IN THE TOTAL AND FINE FRACTIONS OF 10 SOILS FROM EACH STUDY

Sample Fraction[c]	Boston	Baltimore		Cincinnati		GTRI[a]	USDA[b]
	XRF	Hot HNO_3 AAS	Hot HNO_3 ICP	Hot HNO_3 AAS	Cold HNO_3 AAS	XRF	Cold HNO_3 AAS
1T	1,200	1,418	1,324	1,552	1,215	1,174	1,338
2T	1,750	2,893	2,544	2,868	2,211	1,912	2,695
3T	400	492	389	387	466	400	417
4T	550	619	462	423	415	500	464
5T	1,100	1,058	882	964	854	980	988
6T	1,450	2,323	1,955	1,876	1,722	1,524	1,808
7T	1,000	1,359	1,098	1,383	990	651	1,473
8T	500	683	535	491	725	400	726
9T	550	608	485	455	417	261	605
10T	1,450	1,649	1,330	1,679	1,228	1,660	1,764
11T	250	484	365	316	348	180	304
12T	800	1,069	878	1,850	1,103	900	1,944
13T	100		53	63	45	100	73
14T	700	2,200	1,701	2,068	1,713	652	1,710
15T	550	1,754	1,410	747	785	505	825
16T	220	264	200	253	295	187	286
17T	220	126	62	59	58	30	83
18T	75	106	48	74	61	100	111
19T	50	9	7	2	3	20	13
20T	4,800	15,792	12,030	14,593	8,147	4,817	14,733
21T	500	496	372	387	378	383	
22T	950	850	698	837	739	717	1,120
23T	1,700	1,559	1,298	1,567	1,368	1,390	1,761
24T	2,400	2,260	1,880	2,284	2,003	2,021	2,561
26T	2,800	2,484	2,119	2,754	2,401	2,331	2,472
27T	3,800	3,846	3,440	4,337	3,835	3,500	4,983
28T	5,200	5,092	4,667	5,454	4,747	4,460	3,184
29T	4,000	5,097	4,510	5,586	4,700	3,280	6,473
30T	6,500	7,995	6,560	8,467	7,502	4,704	10,042
1F	1,500	1,545	1,421	1,560	1,404	1,223	1,569
2F	2,650	3,540	2,921	3,335	3,127	2,263	3,273
3F	500	625	507	478	508	440	515
4F	1,600	1,814	1,554	1,678	1,595	1234	1,824
5F	1,700	1,793	1,475	1,689	1,971	1,290	1,683
6F	2,400	3,137	2,387	2,835	2,009	2,134	2,682
7F	1,200	1,344	1,105	1,306	1,184	815	1,297
8F	600	723	598	595	298	490	672
9F	650	686	558	593	601	375	630
10F	2,200	2,398	1,946	1,808	1,116	1,980	
11F	220	356	244	267	277	180	280
12F	1,800	2,707	2,220	2,683	2,683	1,680	2,610
13F	100	96	68	68	64	100	89
14F	800	100	779	926	818	693	895
15F	620	796	616	635	642	600	664
16F	300	3,200	236	237	239	236	242
17F	100	118	73	73	66	100	80
18F	100	142	85	91	87	100	92
19F	50		10	3	2	30	20
20F	5,100	7,866	6,000	8,109	7,432	4,780	8,451
21F	550	606	506	480	467	505	470
22F	1,100	1,118	916	1,069	944	980	904

TABLE 3-2 (cont'd). ANALYTICAL RESULTS OF THE FIRST INTERCALIBRATION STUDY: LEAD CONCENTRATION (μg/g) IN THE TOTAL AND FINE FRACTIONS OF 10 SOILS FROM EACH STUDY

Sample Fraction[c]	Boston	Baltimore		Cincinnati		GTRI[a]	USDA[b]
	XRF	Hot HNO_3 AAS	Hot HNO_3 ICP	Hot HNO_3 AAS	Cold HNO_3 AAS	XRF	Cold HNO_3 AAS
23F	1,700	1,679	1,424	1,710	1,431	1,320	1,640
24F	2,200	2,331	2,014	2,328	2,010	1,940	
25F	2,200	2,372	2,000	1,665	2,089	2,005	2,492
26F	2,800	2,899	2,402	2,946	2,568	2,249	3,156
27F	4,000	4,833	3,969	4,531	4,130	3,739	4,979
28F	3,100	3,087	2,616	3,073	2,720	2,445	6,194
29F	4,500	5,896	4,717	5,606	4,869	4,240	6,680
30F	8,000	8,555	7,443	8,679	7,789	6,015	9,754

[a]GTRI = Georgia Tech Research Institute.
[b]USDA = U.S. Department of Agriculture.
[c]T = Total fraction, F = Fine fraction.

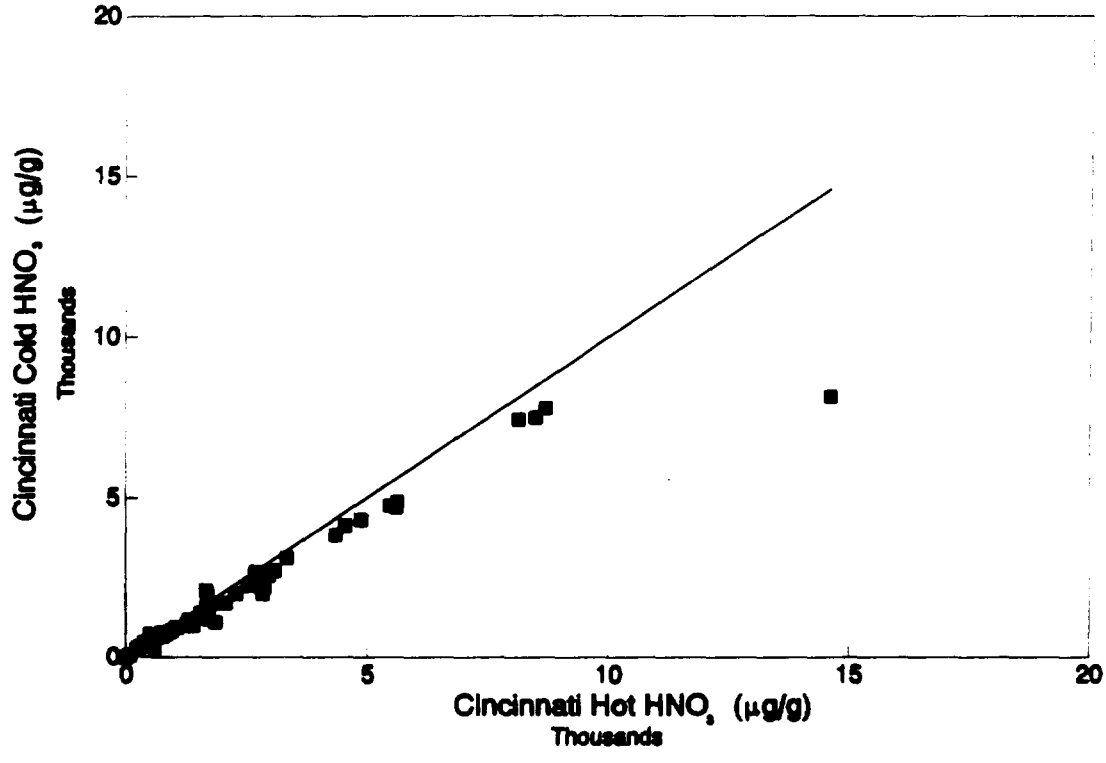

Figure 3-1. Comparison of uncorrected data for two wet chemistry methods of soil analysis showing the comparability of hot and cold nitric acid for the Cincinnati laboratory. The straight line indicates a slope of 1.

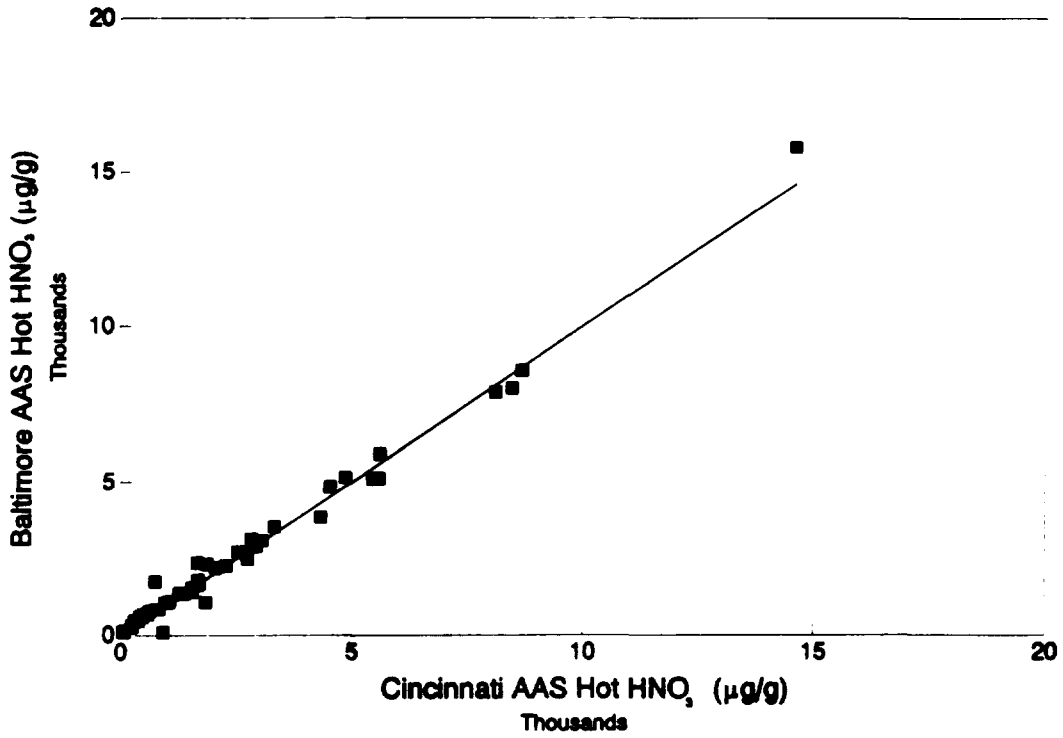

Figure 3-2. Comparison of uncorrected data for atomic absorption spectroscopic analysis by two laboratories (Baltimore and Cincinnati) using the hot nitric acid method of soil analysis. The straight line indicates a slope of 1.

The interlaboratory comparison of XRF between the Boston and GTRI Laboratories showed the method was acceptable, although not fully linear above 5,000 µg/g. There were no soil standards available above 2,000 µg/g, so the analysts had some difficulty calibrating their XRF instruments above this level. The data shown in Figure 3-3 suggest a systematic difference between the two laboratories that could be corrected with a more uniform calibration. Both interlaboratory (Cincinnati and Baltimore in Figure 3-4) and intralaboratory (Baltimore in Figure 3-5) comparisons of AAS versus ICP demonstrated equivalency between these two instrumental methods. These comparisons showed that there is likewise a systematic difference that can be statistically corrected.

Finally, the interlaboratory comparison of XRF versus AAS (Boston and Cincinnati in Figure 3-6, and Boston and Baltimore in Figure 3-7) led to the conclusion that, if suitable soil standards at higher concentrations could be made available, XRF would be an acceptable alternative method to AAS for soil analysis.

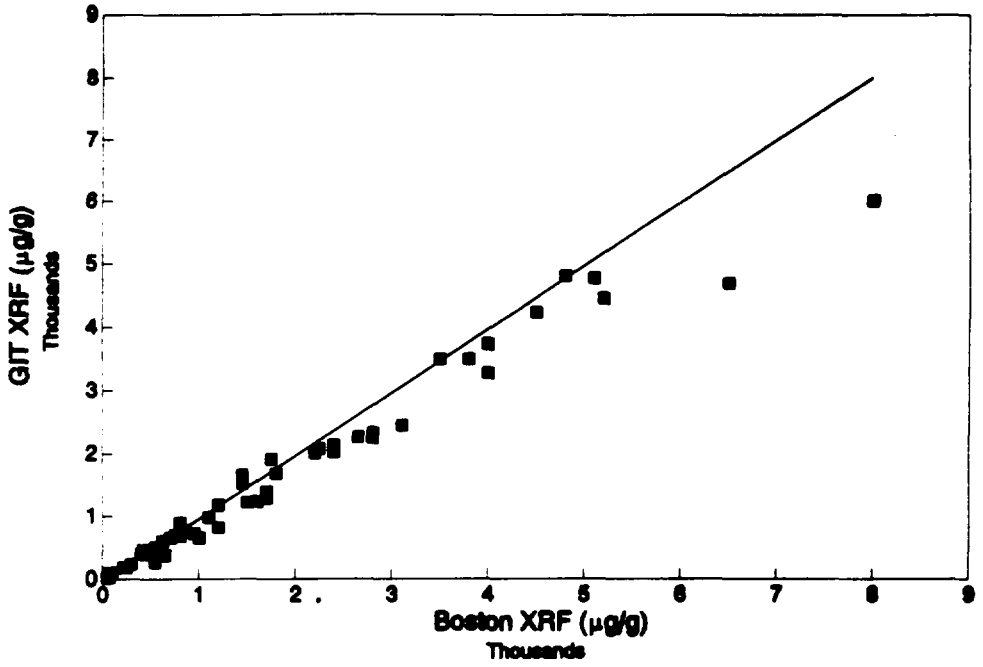

Figure 3-3. Interlaboratory comparison of uncorrected data for the X-ray fluorescence method of soil analysis showing the comparability of the Boston and Georgia Institute of Technology laboratories. The straight line indicates a slope of 1.

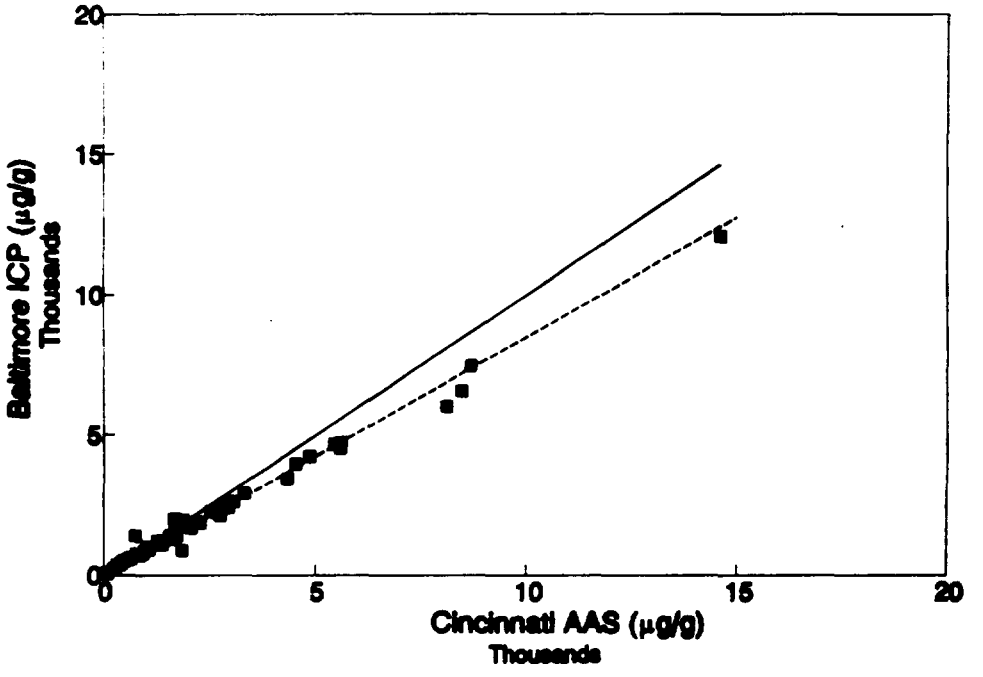

Figure 3-4. Interlaboratory comparison of uncorrected data for soil analysis showing the comparability of inductively coupled plasma emission spectroscopy and atomic absorption spectroscopy for the Baltimore and Cincinnati laboratories. The straight line indicates a slope of 1.

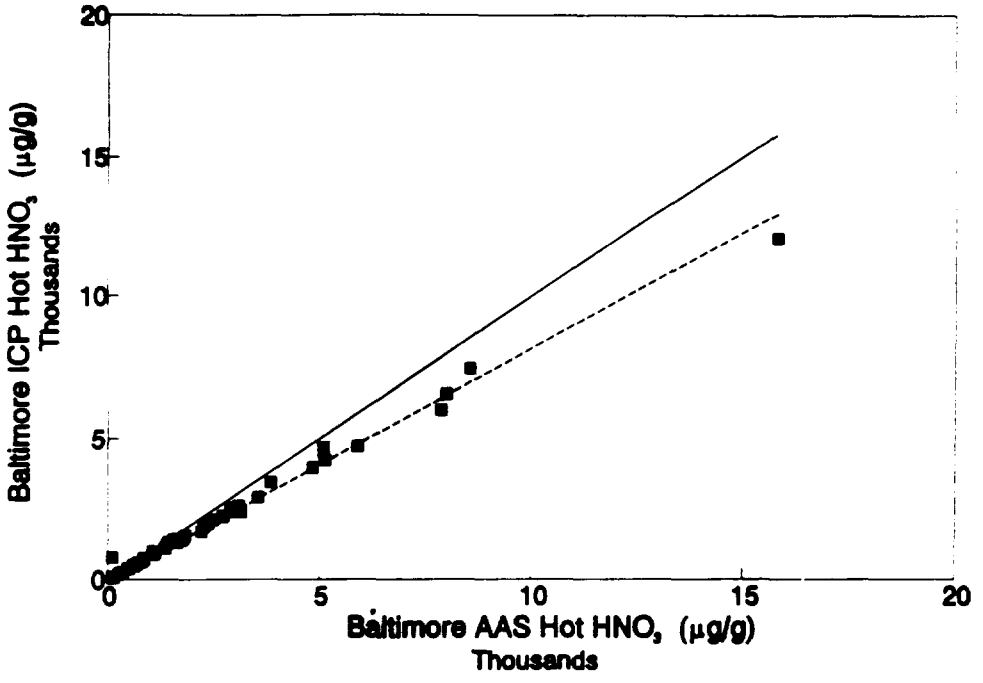

Figure 3-5. Comparison of uncorrected data for soil analysis showing the comparability of inductively coupled plasma emission spectroscopy and atomic absorption spectroscopy within the Baltimore laboratory. The straight line indicates a slope of 1.

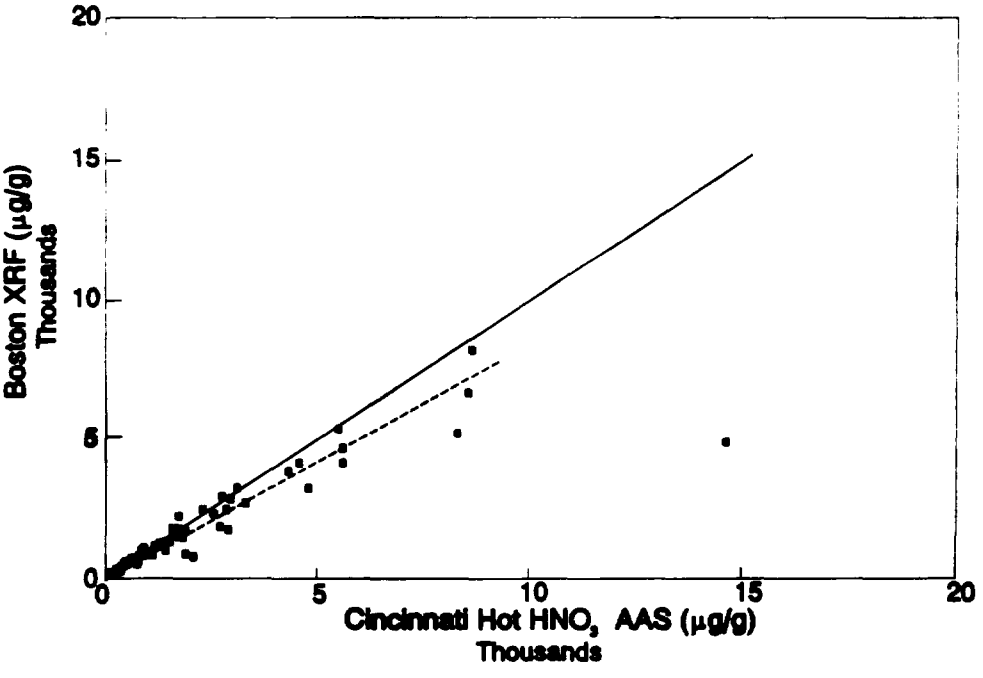

Figure 3-6. Interlaboratory comparison of uncorrected data for soil analysis showing the comparability of X-ray fluorescence and atomic absorption spectroscopy for the Cincinnati and Boston laboratories. The straight line indicates a slope of 1.

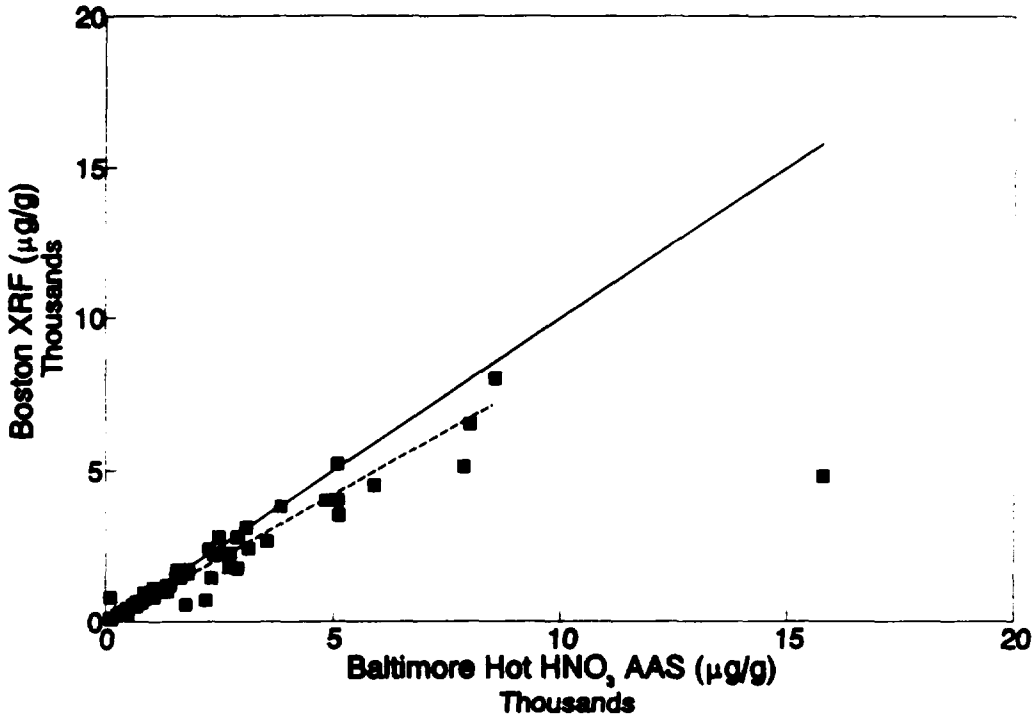

Figure 3-7. Interlaboratory comparison of uncorrected data for soil analysis showing the comparability of X-ray fluorescence and atomic absorption spectroscopy for the Baltimore and Boston laboratories. The straight line indicates a slope of 1.

The Scientific Coordinating Panel recommended the use of XRF for soil analysis on the condition that a suitable set of common standards could be prepared for a broader concentration range and that a rigorous audit program be established to ensure continued analytical accuracy. This recommendation was based on the interlaboratory comparison study, the awareness that chemical extraction of a large number of soil samples presented a costly burden on the project both in terms of time and expense, and the value of nondestructive analysis in preserving the samples for reanalysis. The Round Robin I calibration exercise also revealed the need for a broader scale calibration exercise to determine the arithmetic correction factor for converting the data to a common basis.

Two groups, Boston and Baltimore, also elected to use XRF for interior dust analysis, whereas Cincinnati opted for hot nitric extraction with AAS for interior dust and XRF for exterior dust. During the study, Baltimore recognized problems with analyzing dust by XRF when the sample size was small (less than 100 mg). They reanalyzed the dust samples by

AAS and reported both measurements. In Boston, this problem was solved by compositing the floor dust samples for XRF analysis, reporting one floor dust sample per housing unit.

3.1.2 Quality Assurance/Quality Control Standards and Audits

After the first intercalibration exercise, a set of nine soil and six dust interlaboratory standards was prepared to monitor the QA/QC performance of soil and dust analysis throughout the project. These were prepared from three soil and two dust samples from each of the three studies, collected in bulk (about 30 kg), in a range thought to be high, medium, and low for that area. Seven of the soil samples and five of the dust samples were dried, sieved, and analyzed at the EPA Environmental Monitoring Systems Laboratory in Las Vegas, NV (EMSL/LV). Following homogenization, approximately 50 aliquots of each of the samples were analyzed by laboratory scale XRF at the EMSL/LV laboratory to estimate the acceptable range for a single laboratory. Three of the 15 were distributed to the participating cities for use as interlaboratory reference standards. The remaining 12 were used as double blind external audits for soil and dust.

Each city appointed a QA/QC officer who was not directly involved with the analysis of the soil samples, but who had access to the soil sample preparation stream on a daily basis. This person mailed prelabeled soil sample containers with typical sample numbers to the EMSL/LV laboratory. Approximately 20 g samples from one of the six external audit materials typical for each city were placed in the sample containers fully disguised as field soil samples and returned to the QA/QC officer in lots of 20 to 30. The identification numbers and soil concentration values were monitored by the project QA/QC officer at ECAO/RTP. Each city's QA/QC officer inserted the double blind samples into the sample stream on a random basis at a frequency that would ensure about four QA/QC samples per analytical day. These were occasionally placed as duplicates in the same batch to provide information about replication within the batch.

The preliminary acceptance range for the double blind audit samples was established using the original 50 XRF analyses by the Las Vegas laboratory discussed above. As the analytical results were reviewed by the study QA/QC officer, the audit sample results were sent to the project QA/QC officer at ECAO/RTP. If the audit samples were outside the acceptable range, the study QA/QC officer was informed and could recommend either

reanalysis or flagging the data for that entire batch. The initial acceptable range for the six audit samples was based on analyses by a single laboratory (EMSL/LV). This range was adjusted for interlaboratory variation after the Intercalibration Exercise II. Final decisions on the disposition of the audit sample anomalies were deferred until the completion of the second intercalibration exercise near the end of the study.

The results of the double-blind audit program are given in Table 3-3 based on the final biweight distributions shown in Table 3-4. The preliminary biweight distributions, shown also in Table 3-4, contained no measure of interlaboratory variability because the preliminary analyses were performed by only the EMSL-LV laboratory. These values could only be used in a preliminary assessment of the audit program to identify and flag batches of soil samples that might need to be reanalyzed pending the determination of the final biweight distributions.

The laboratories were found to be systematically low or high. This was not of major concern, as these discrepancies could be resolved by a more detailed intercalibration exercise and statistical correction at the end of the study. The Cincinnati group elected to make a midcourse change in instrumental parameters that reduced this difference, and they described this procedure in their report. Occasionally, the measured audit sample was sporadically high or low, in which case the laboratory investigated the problem and resolved it. Most of these discrepancies occurred for dust samples where the sample size for XRF analysis was below 200 mg. The Boston group found, but did not report in detail, that a calibration curve for XRF analysis using standards that were also less than 200 mg would provide a suitable correction to the original data. They elected, however, to composite their floor dust samples.

3.1.3 Round Robin Intercalibration Exercise II

Near the end of the project, aliquots of the nine soil and six dust audit samples used during the project were redistributed to the three study laboratories for single blind analysis. The analyst was aware that the samples were audit samples, but did not know their concentrations. These measurements were the basis for establishing the final range of acceptability for the audit samples and for adjusting the soil and dust measurements in each study to values common to the project.

TABLE 3-3. SOIL AND DUST AUDIT PROGRAM RESULTS

Study/Audit Sample	Number of Samples	Mean (μg/g)	Range (μg/g)	Percent Within Final Biweight Distribution[a]
BOSTON DUST (XRF)				
BAL 03	N/A[b]	1,232	980-1,441	92
CIN 01	N/A	2,671	2,075-3,228	100
CIN 02	N/A	331	115-461	65
BOSTON SOIL (XRF)				
BOS M	N/A	6,786	6,015-7,549	100
BAL H	N/A	1,044	747-1,244	73
CIN L	N/A	399	207-570	61
CIN H	N/A	14,074	11,407-16,592	50
BALTIMORE DUST (XRF)				
BAL 02	8	218	159-281	100
CIN 01	10	3,280	800-3,660	90
BOS 01	10	14,444	14,080-14,920	N/A
BALTIMORE SOIL (XRF)				
BOS M	15	5,046	4,800-5,200	100
BAL H	15	838	433-916	60
CIN L	15	286	266-307	100
CIN H	15	11,290	10,100-12,500	53
CINCINNATI DUST (AAS)				
BAL 03	34	1,727	1,322-2,687	N/A
BOS 01	35	24,104	20,266-27,962	N/A
CIN 01	38	2,683	2,070-3,163	100
CIN 02	26	259	200-393	N/A
CINCINNATI SOIL (XRF)				
BOS M	32	5,580	4,759-6,107	100
BAL H	49	885	822-1,012	N/A
CIN L	130	263	244-310	100
CIN H	31	12,304	9,838-13,632	N/A

[a]These percentages include audit samples for which analyses were outside the biweight distribution range and for which the action required by the QA/QC plan, such as reanalysis of the entire batch, was implemented.
[b]N/A = Not available.

TABLE 3-4. PRELIMINARY AND FINAL BIWEIGHT DISTRIBUTIONS FOR SOIL AND DUST AUDIT PROGRAM

Sample Type	Audit Sample	Preliminary Values (μg/g)			Final Values (μg/g)		
		Mean	Low	High	Mean	Low	High
Dust	BAL01	78	58	99	84	4	163
Dust	BAL02	331	288	374	309	138	480
Dust	BAL03	1,480	1,346	1,613	1,438	1,091	1,786
Dust	CIN01	2,851	2,660	3,042	2,617	1,422	3,812
Dust	CIN02	252	216	288	233	93	372
Soil	BOS L	3,131	2,858	3,405	3,101	2,283	3,919
Soil	BOS M	6,090	5,748	6,431	6,219	4,742	7,696
Soil	BOS H	14,483	13,071	15,895	13,369	11,980	14,754
Soil	BAL L	639	555	724	626	468	783
Soil	BAL H	923	850	997	1,017	847	1,187
Soil	CIN L	303	284	322	315	204	426
Soil	CIN H	13,585	12,872	14,297	12,729	11,361	14,096
Soil	REF5				413	258	568
Soil	REF6				936	738	1,134
Soil	REF7				1,042	758	1,326
Soil	REF8				2,354	1,950	2,759
Soil	REF9				3,913	2,943	4,888
Soil	REF10				735	615	854

3.1.4 Biweight Distribution and Final Interlaboratory Calibration

The nine soil and five dust samples that were used for external standards and audit samples were reanalyzed in a more detailed round robin exercise near the end of the project. The purpose of this exercise was to determine the correction factor for statistically converting the soil and dust data from each study to a common basis and to revise the biweight distribution values for the audit samples to reflect the multilaboratory variance and systematic differences between laboratories. Additional analyses by AAS were performed by Baltimore

and Cincinnati for soil and dust, even though only dust was analyzed by AAS during the study. Boston and Las Vegas analyzed the samples by ICP for the purposes of obtaining a broader perspective on the application of this method. The data from this exercise are shown in Table 3-5 and are the basis for determining the consensus values and correction factors that appear in Table 3-6.

A data evaluation subcommittee of the Scientific Coordinating Panel was appointed to determine the consensus values and methods of statistical interpretation of the intercalibration results. Several methods were discussed in great detail. Tests were made for outliers using the method of Barnett and Lewis (1984), and none were found. The data were of good quality and were highly linear. The r^2 values ranged from 0.997 to 0.999 using a consensus based on the simple arithmetic means of the reported values. The subcommittee chose to explore alternatives to the arithmetic mean and eventually settled on a multiplicative model weighted for within-laboratory variance. The model was run with GLIM statistical software, Version 3.77, Update 2, and gave consensus values and correction factors as shown in Table 3-6. Although several alternatives to simple regression were evaluated, the consensus values produced by the GLIM procedure differed only slightly from those of a simple linear regression. The correction factors in Table 3-6 were used by the three studies to convert their soil and dust data to a common project basis. A plot of the dust (Figure 3-8) and soil (Figure 3-9) reported values versus the consensus means derived from the GLIM analysis illustrates the reliability of this method.

3.1.5 Disposition of Audit Data

Based on the results of the second intercalibration exercise, a consensus value was determined for each dust and soil sample, and biweight distributions were determined for those that had been used in the audit program. This new distribution incorporated interlaboratory variation. When the correction factor is applied to the reported results, the revised number should lie between the upper and lower boundaries of the biweight distribution. Table 3-3 lists the percentage of these audit sample values that fell within these new boundaries. Most of the discrepancies were resolved by the corrective measures taken by the laboratories.

TABLE 3-5. RESULTS OF THE FINAL INTERCALIBRATION STUDY (μg/g)

	XRF					AAS		ICP	
Sample	BOSK	BOSX	BAL	CIN	LV	BAL	CIN	BOS	LV
DUST1	120		121	92	78	15	66	94	72
DUST2	320		482	329	288	201	236	284	307
DUST3	1,430		1,686	1,307	1,288	1,363	1,581	1,428	1,346
DUST4	2,000		3,771	2,924	2,456	2,335	2,451	2,109	2,296
DUST5	280		267	233	212	150	273	244	191
SOIL1	450	510	388	441	310	383	452	401	379
SOIL2	900	910	808	1,033	833	1,001	1,013	850	912
SOIL3	1,050	1,100	961	1,080	923	1,100	1,120	972	1,006
SOIL4	2,200	2,300	2,100	2,555	2,264	2,468	2,502	2,230	2,286
SOIL5	3,800	4,000	3,486	4,227	3,974	4,044	4,251	3,748	3,843
SOIL6	710	770	640	789	611	741	798	699	660
SOIL7	650	930	559	675	532	567	650	597	626
SOIL8	950	930	896	1,036	798	1,032	1,067	944	998
SOIL9	2,800	2,900	2,514	3,126	2,972	3,401	3,263	3,148	3,158
SOIL10	5,600	5,300	5,200	6,493	5,956	6,861	6,937	5,932	6,360
SOIL11	12,500	13,000	11,000	15,963	15,984	13,175	13,955	12,652	12,608
SOIL12	310	290	283	305	286	321	379	300	294
SOIL13	12,000	12,000	10,500	14,156	13,530	13,000	13,195	13,167	11,440
SOIL14	810	850	793	929	763	875	986	907	900
SOIL15	1,450	1,600	1,400	1,705	1,509	1,731	1,766	1,631	1,650

When the audit sample values fell outside the boundaries of the final biweight distribution, the batches were flagged. The options could then be to exclude these data from the statistical analysis, reanalyze the samples, or use the original data based on other evidence that the data are correct. The quality of soil and dust analysis in this project was equal to or greater than the generally acceptable standards for reporting soil and dust data in the scientific literature.

TABLE 3-6. CONSENSUS VALUES AND CORRECTION FACTORS FROM THE FINAL INTERCALIBRATION PROGRAM

	XRF	AAS	ICP
Interlaboratory Consensus Values for Dust (μg/g)			
Sample			
DUST1	92.8	54.2	81.7
DUST2	342.7	221.9	283.4
DUST3	1,319.0	1,492.2	1,362.3
DUST4	2,943.4	2,378.1	2,133.4
DUST5	228.3	232.4	206.2
Interlaboratory Correction Factors for Dust[a]			
Study			
BOS	1.1527		1.0707
BAL	0.7803	1.0416	
CIN	1.0074	0.9616	
Interlaboratory Consensus Values for Soil (μg/g)			
Sample			
SOIL1	460.2	430.5	426.6
SOIL2	960.7	1,002.1	909.6
SOIL3	1,140.5	1,106.2	1,018.8
SOIL4	2,493.5	2,474.2	2,342.1
SOIL5	4,139.3	4,164.1	3,706.1
SOIL6	761.0	776.9	736.1
SOIL7	664.1	623.3	656.0
SOIL8	1,062.3	1,049.4	1,005.4
SOIL9	2,987.8	3,272.6	3,274.9
SOIL10	6,175.2	6,863.2	6,411.5
SOIL11	13,120.7	13,645.4	13,224.7
SOIL12	335.3	361.5	323.6
SOIL13	12,498.5	13,041.6	13,080.0
SOIL14	941.3	949.5	923.3
SOIL15	1,663.2	1,744.1	1,716.8
Interlaboratory Correction Factors for Soil[a]			
Study			
BOS	1.0370		1.0166
BAL	1.1909	1.0166	
CIN	0.8698	0.9839	

[a] The correction factor is the value that the reported soil or dust measurement should be multiplied by in order to adjust each value to a common basis among all three studies.

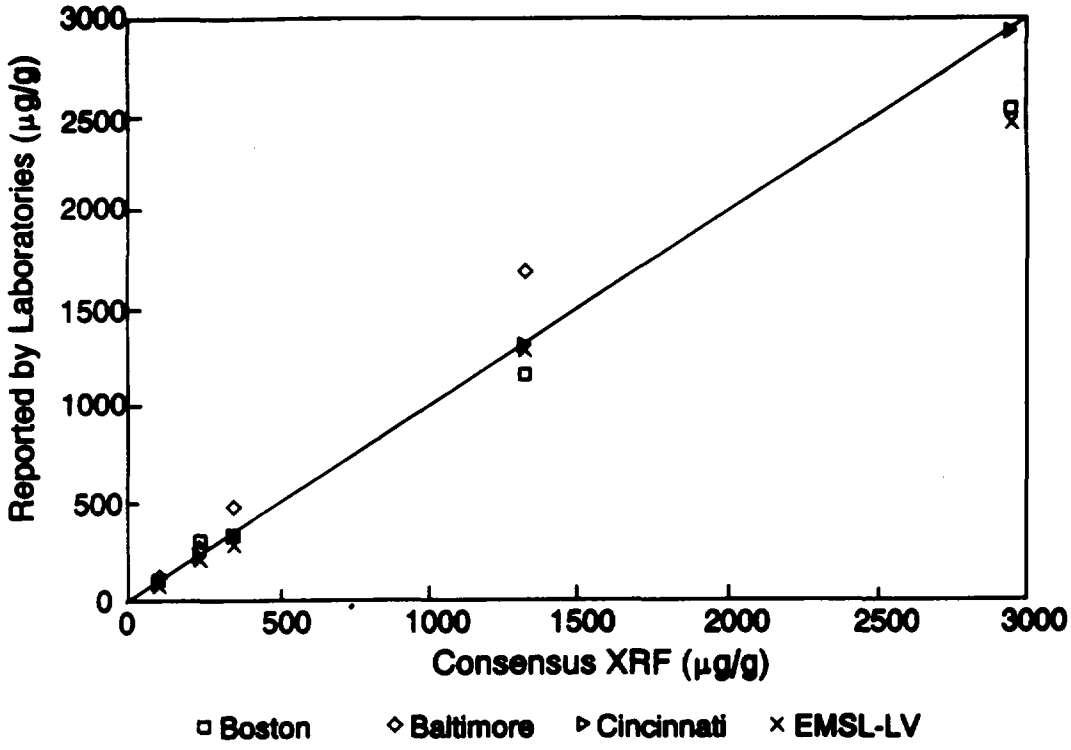

Figure 3-8. Departures from consensus dust values for each of the three studies.

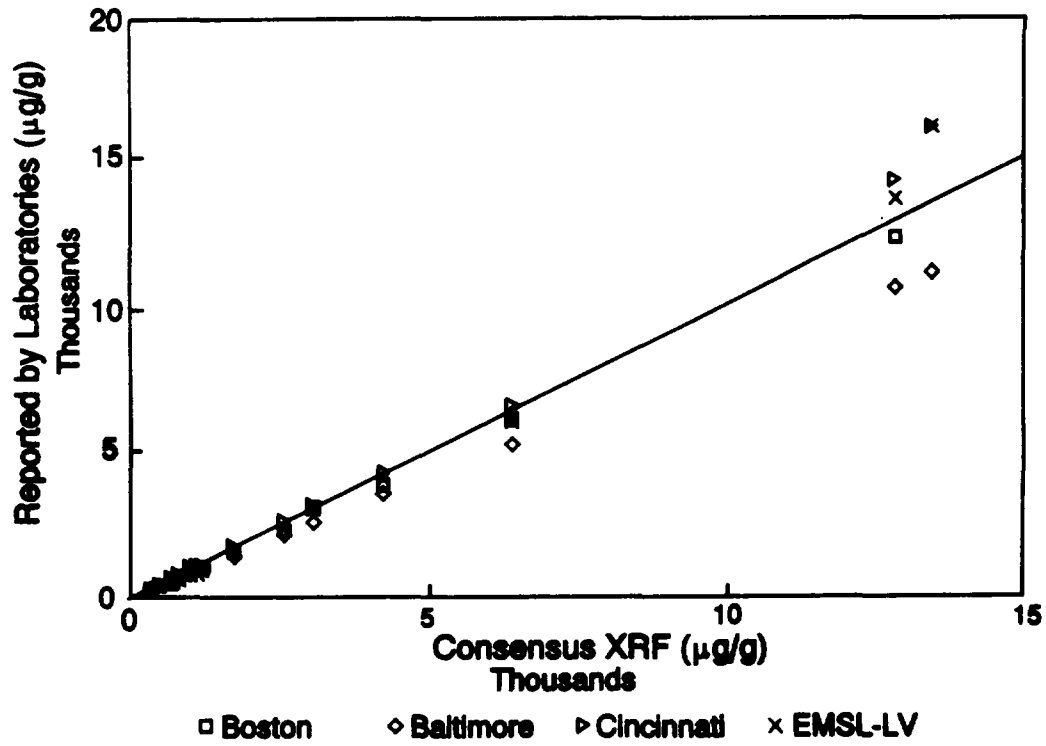

Figure 3-9. Departures from consensus soil values for each of the three studies.

3.2 QUALITY ASSURANCE AND QUALITY CONTROL FOR HAND DUST

The collection and analysis of hand wipes is an innovative procedure developed just prior to the beginning of the project. There were few published reports of the measurement techniques, no certified standards, no internal standards, and little information on which to base decisions for acceptable analytical precision. Double blind audit samples were provided to the study QA/QC officer as an external control for hand wipe analysis. These were prepared as simulated samples by placing a known amount of an appropriate solution of lead nitrate onto the blank hand wipe at the EMSL/LV laboratory, wrapping and labeling according to the field protocol and returning to the participating laboratory for insertion into the sample scheme. There was no attempt to determine interlaboratory variance or to calculate correction factors. The study QA/QC officer was responsible for reporting problems to the laboratory director.

3.3 QUALITY ASSURANCE AND QUALITY CONTROL FOR BLOOD LEAD

The QA/QC program for blood analysis was directed by Dr. Dan Paschal of the Centers for Disease Control and Prevention (CDC), using the protocols developed for the CDC blood lead certification program. Each laboratory received double blind bovine blood samples from CDC Blind Pool 1 and Blind Pool 2 and inserted these blind samples into the blood sample stream for the duration of the study. The data from this QA/QC program are shown in Table 3-7. These data show the number of exceedances to be zero for all three studies. An exceedance occurs when the mean of two replicates exceeds the 99th percentile range established by CDC. The data also allow estimation of the probability of analytical drift during the period of analysis. There was evidence for drift in the Boston Blind Pool 2 and marginal evidence in Cincinnati Blind Pool 1. While the statistical analysis of the QC data for Boston blood lead analyses suggest the possibility of analytical drift (of unknown direction) for part of the period where blood lead data were being sampled and analyzed, the statistical methods for evaluating abatement effectiveness used by the investigators and by this assessment would compensate for any possible analytical drift.

TABLE 3-7. QUALITY CONTROL RESULTS FOR CENTERS FOR DISEASE CONTROL AND PREVENTION BLIND POOL BLOOD LEAD ANALYSES

		Blind Pool 1				Blind Pool 2			
Study	Dates	n	Number of Exceedances[1]	Target Range[2]	Drift[3]	n	Number of Exceedances[1]	Target Range[2]	Drift[3]
Boston	Jul 89 - Aug 91	123	0	1.9-6.1	0.2092	112	0	8.0-13.1	0.0389
Baltimore	Aug 88 - Oct 90	66	0	3.9-6.2	0.6382	59	0	9.2-13.1	0.4748
Cincinnati	Aug 88 - Oct 90	53	0	1.4-5.6	0.0672	48	0	6.5-11.2	0.4732

[1]Number of samples that exceeded the target range established by CDC for each batch of QC blood analyses within a pool.

[2]The target range is the upper and lower 99th percentile confidence limit established by CDC and differs for each Blind Pool and each method of analysis.

[3]The drift test probability is a P-value for the test of the hypothesis that the slope of the difference between the reported values and the CDC accepted value is significantly greater than zero. A P-value less than 0.05 indicates this slope may be greater than zero and that some analytical drift may have occurred over time, but the direction of this possible drift is not indicated by this statistic.

3.4 DATABASE QUALITY

Each study maintained rigorous standards for database quality. These included double entry, 100% visual confirmation, and standard statistical procedures for detecting outliers.

In reviewing the data for statistical analyses contained in this Integrated Report, some data ambiguities or errors were found, confirmed, and corrected prior to use in this assessment. None of these, however, would have impacted the conclusions drawn by the individual study reports.

This evaluation of the QA/QC data shows that the three studies were comparable in their ability to meet the requirements of their QA/QC program. Furthermore, their performance on the audit program and intercalibration exercises suggests that the data are comparable among the three studies, with the appropriate correction factors shown in Table 3-6.

4. INDIVIDUAL STUDIES

4.1 INDIVIDUAL STUDY INTERVENTION STRATEGIES AND SAMPLING PLANS

4.1.1 Boston Study

The pathway intervention scheme for Boston is shown in Figure 4-1. In Boston, all properties in the soil abatement group were abated. To be eligible to participate in the study, the average or median soil lead concentration was greater than 1500 μg/g. The approach to soil abatement was to remove the top 15 cm of soil, apply a synthetic fabric, and cover with a layer of about 20 cm of clean topsoil. The new soil was covered with sod or seeded with grass and watered through dry months. Areas not seeded or resodded were covered with a bark mulch. Some driveways and walkways were covered with 5 cm soil and 15 cm gravel or crushed bank (stone with dust). On four properties, the driveway and yard were capped with 7.5 cm asphalt without soil removal, at the owner's request. A total of 93 Boston properties, including those abated at the end of the project, were abated in this manner. The information on area treated and volume of soil removed from these properties appears in Table 4-1. The method of excavation was by small mechanical loader (Bobcat) and hand labor, for the most part. Initially, six properties were abated with a large vacuum device mounted on a truck, but this proved to be unsatisfactory due to the size and lack of maneuverability. During one extreme cold spell, it was necessary to remove large blocks of frozen soil, often greater than 15 cm thick, by loosening with a jackhammer.

In Boston, loose paint stabilization consisted of removing chipping and peeling paint with a HEPA vacuum and washing the surfaces with a trisodium phosphate and water solution. Window wells were painted with a fresh coat of primer.

Interior dust abatement was performed after loose paint stabilization. Families spent the day off-site during interior dust abatement. Hard surfaces (floors, woodwork, window wells, and some furniture) were vacuumed with a High-Efficiency Particle Accumulator (HEPA) vacuum, as were soft surfaces such as rugs and upholstered furniture. Hard surfaces were also wiped with a wet cloth (an oil treated rag was used on furniture) following vacuuming. Common entries and stairways outside the apartment were not abated.

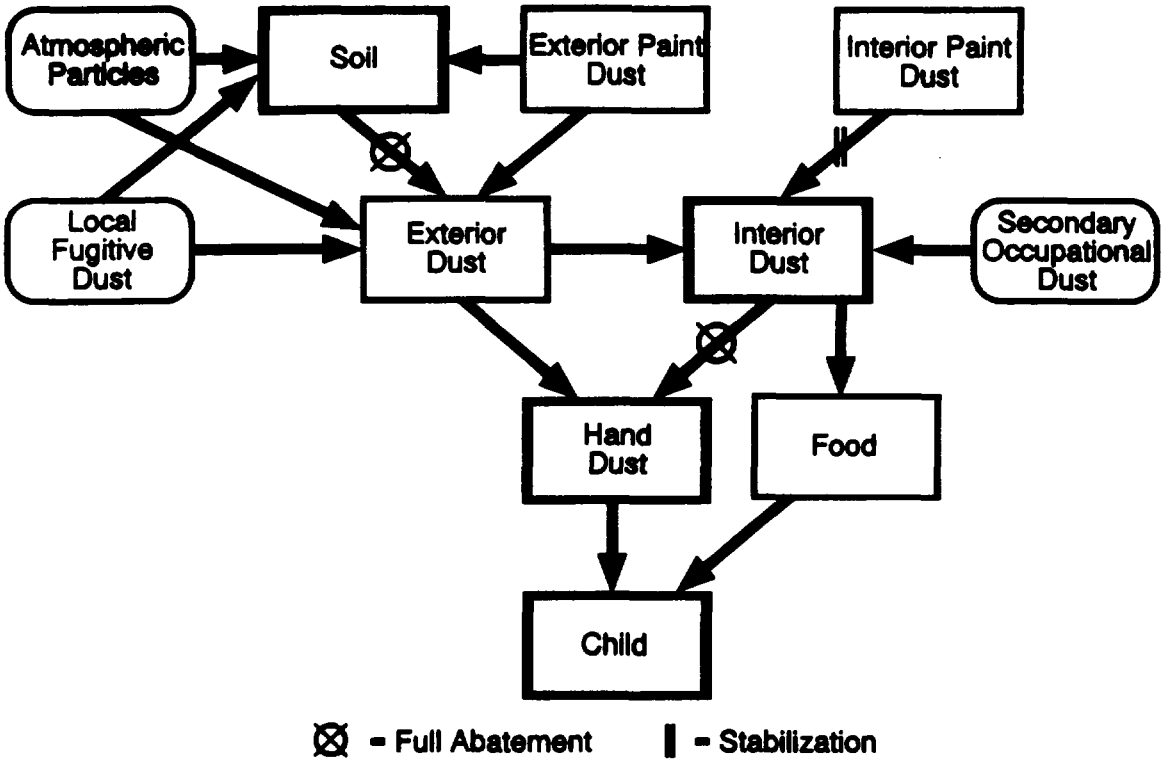

Figure 4-1. Pathway intervention scheme for dust exposure (Boston Soil Abatement Study). Bold-line rectangles indicate pathway components monitored by sequential sampling.

TABLE 4-1. SOIL ABATEMENT STATISTICS FOR THE THREE STUDIES

	Boston	Baltimore	Cincinnati
Number of properties[a]	36	63	171
Surface area (m^2)	7,198	4,100[b]	12,089
Volume soil removed (m^3)	1,212	690	1,813
Surface area/property (m^2)	200	73	71
Volume soil/property (m^3)	34	11	11

[a]Includes only properties abated during study. Properties abated at the end of the study, where no further sampling was reported, are not included in this analysis, but are included in the individual study reports. In Cincinnati, a property is the location of the soil abatement, not the location of the child's residence.
[b]Surface area not provided by Baltimore report. This was calculated using Boston volume-to-surface ratio, which is equivalent to an average removal depth of 17 cm.

Although subsequent measurements of lead-based paint were made, no measurements were made of the movement of lead from paint to house dust that would reflect the effectiveness or persistency of paint stabilization. It was believed that any contamination from lead-based paint would be readily apparent in the dust samples.

Between Rounds 1 and 3, the Boston study lost only three of the original 152 children enrolled. Twenty-two of the children moved to a new location but were retained in the study through followup and analysis of their new residence. Children with blood lead concentrations below 7 μg/dL or above 24 μg/dL had been excluded from the study and two of the children were dropped from the data analysis when they developed lead poisoning, probably due to exposure to lead-based paint away from their home.

Baseline characteristics (age, SES as derived from the Hollingshead Index, soil lead, dust lead, drinking water lead, and paint lead) were similar for the three Boston study groups (BOS P-S, BOS PI-S, BOS SPI). The preabatement blood lead concentration was higher for BOS P-S. The proportion of Hispanics was higher in BOS P-S than in BOS PI-S or BOS SPI, and the proportion of Blacks was lower. There was a larger proportion of male children in BOS P-S.

Data were analyzed by comparison of group means using analysis of covariance (ANCOVA), which showed a significant effect of group assignment (intervention) for both the BOS PI-S and BOS SPI groups. These results did not change with age, sex, socioeconomic status, or any other variable except race and paint loading (P-XRF measurement). When blood lead was adjusted for paint lead loading, the effect of the soil abatement relative to the two control groups was somewhat smaller and had a lower statistical significance ($P = 0.06$ versus $P = 0.02$). Likewise, adjusting blood lead for race reduced the size and statistical significance of the effect of soil abatement ($P = 0.09$ versus $P = 0.02$).

The Boston study has some limitations. Participants were chosen to be representative of the population of urban preschool children who were already at risk of lead exposure. The Boston Childhood Lead Poisoning Prevention Program was used to identify potential participants from neighborhoods with the highest rates of lead poisoning. Because no study subjects had blood lead levels below 7 μg/dL or in excess of 24 μg/dL at baseline,

extrapolation of the effect of lead contaminated soil abatement for children above or below this range is difficult.

Follow-up blood lead measurements were made in Boston eleven months after intervention and again at 23 months.

4.1.2 Baltimore Study

In Baltimore, 63 properties in BAL SP were abated between August and November 1990. An additional seven properties that did not meet the requirements for abatement were transferred to the control group (BAL P2). The pathway intervention scheme is shown in Figure 4-2. Soil covered areas on each property were divided into segments or parcels, usually front, back, and side. Any parcel with soil lead concentrations above 500 μg/g was abated entirely. Soil and ground cover were removed down to 15 cm and replaced to the original level with soil having a lead concentration less than 50 μg/g. These areas were sodded or reseeded as appropriate. Bare areas were prepped and reseeded even if soil lead concentrations did not warrant excavation. Additional abatement statistics appear in Table 4-1.

The exterior painted surfaces of Baltimore homes were wet scraped over the chipping and peeling surfaces, followed by HEPA vacuuming. The entire surface was primed and painted with two coats of latex paint.

The Baltimore study recruited 472 children, of whom 185 completed the study. Of those that completed the study, none were excluded from analysis. The recruited children were from two neighborhoods, originally intended to be a treatment and a control group. Because soil concentrations were lower than expected, some properties in the treatment group did not receive soil abatement. In their analysis, the Baltimore group transferred these properties to the control group.

Because of logistical problems, there was an extended delay between recruitment and soil abatement that accounted for most of the attrition of the participating families from the study. In their report, the Baltimore group applied several statistical models to the two populations to evaluate the potential bias from loss of participating children. These analyses showed that the two populations remained virtually identical in demographic, biological and environmental characteristics.

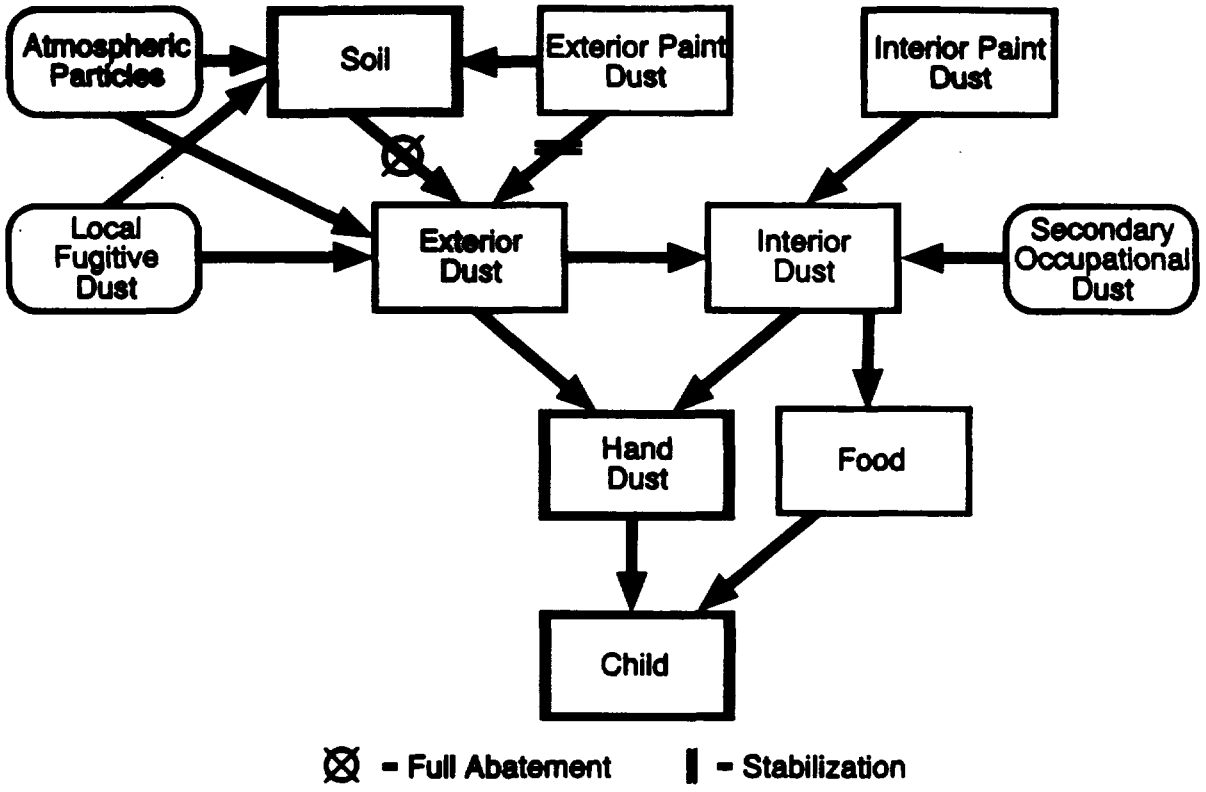

Figure 4-2. Pathway intervention scheme for dust exposure (Baltimore Soil Abatement Study). Bold-line rectangles indicate pathway components monitored by sequential sampling.

The Baltimore study design focused on changes in biological parameters (hand dust and blood lead) over an extended period of time. The study provided limited information on changes in the movement of lead in the child's environment in response to intervention. Repeat measurements of soil were done for abated properties only, to confirm abatement. There were no abatement measurements of exterior dust, no interior paint stabilization, and no interior dust abatement.

Including the prestudy screening measurements of hand dust and blood lead in the original cohort of participants, the Baltimore study made six rounds of biological measurements that spanned twenty months.

4.1.3 Cincinnati Study

The pathway scheme for the Cincinnati study is shown in Figure 4-3. Within each of six neighborhoods, the Cincinnati study identified all sites with soil cover as discrete soil parcels. The decision to abate was based on soil lead concentrations for each parcel of land, and for the depth to which the lead had penetrated. Lead was measured at two depths, the top 2 cm and from 13 to 15 cm. If the average concentration of the top and bottom samples was 500 μg/g or greater, the soil was removed and replaced, regardless of the adequacy of the top cover. If any of the top two cm composite samples exceeded 500 μg/g, that parcel was also abated. Initially, there was an option to cultivate by roto-tilling, but this approach was abandoned as not feasible in this study. For areas where the top concentration was greater than or equal to 300 μg/g, and the average concentration of the top and bottom samples was less than 500 μg/g and the cover was inadequate, the soil was resodded. Excavation was by front end loader, backhoe, and hand tools down to 15 cm, and the replacement soil lead concentration was less than 50 μg/g. Further abatement statistics can be found in Table 4-1.

The approach to exterior dust abatement was to identify all parcels with one of several types of exterior hard surfaces in the neighborhood where dust might collect, to obtain permission to sample and abate these areas, and to clean them once with vacuum equipment suitable for the parcel. This vacuum equipment had previously been tested and shown to remove about 95% of the available dust on the area. The types of surfaces identified were streets, alleys, sidewalks, parking lots, steps, and porches. For data analysis in the Cincinnati report, these were grouped as (1) targeted areas adjacent to the exterior of the buildings where children lived, such as steps, porches, and sidewalks; (2) streets, sidewalks, and alleys throughout the study neighborhoods; and (3) parking lots and other paved areas throughout the study neighborhoods.

The exterior dust measurements in the Cincinnati study (and the interior dust measurements of all three studies) were made in a manner that determined the lead concentration (μg Pb/g dust), the dust loading (mg dust/m^2), and the lead loading (μg Pb/m^2) for the surface measured. This required that a dry vacuum sample be taken over a prescribed area, usually 0.25 to 0.5 m^2. It is important to note that dust abatement is

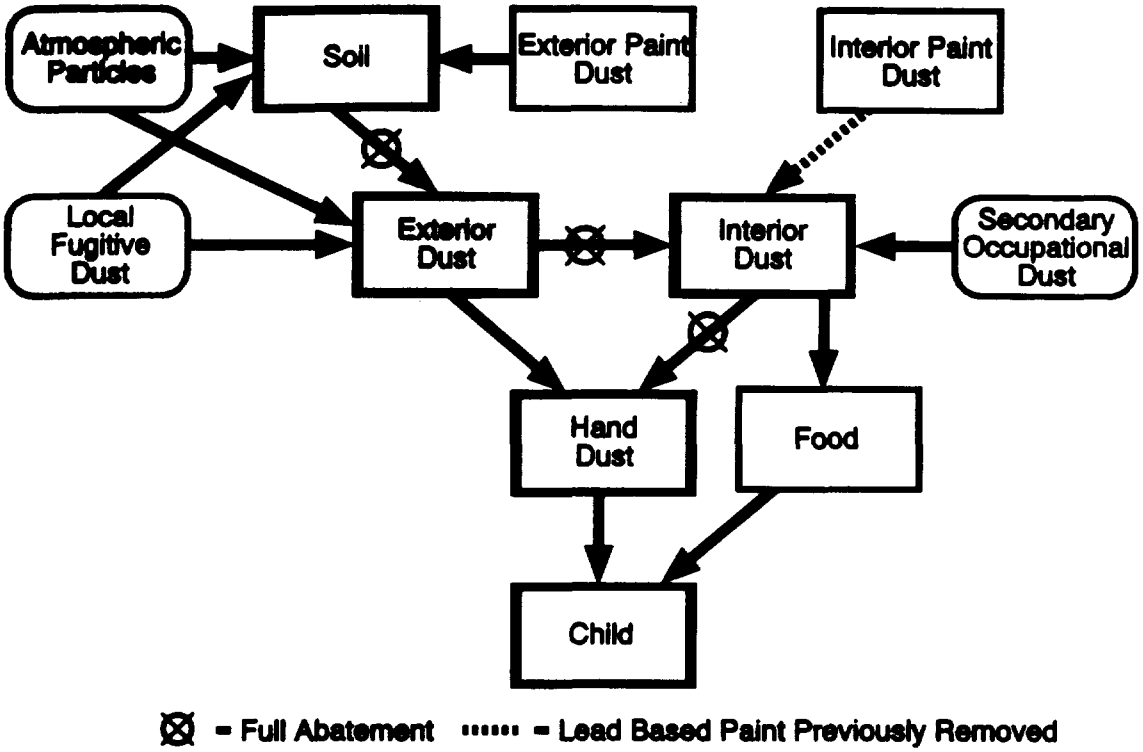

Figure 4-3. Pathway intervention scheme for dust exposure (Cincinnati Soil Abatement Study). Bold-line rectangles indicate pathway components monitored by sequential sampling.

expected to cause an immediate change in the dust and lead loading, but not necessarily in the lead concentration on dust surfaces.

The Cincinnati group performed interior dust abatement after exterior dust abatement, moving the families off-site during this activity. Vacuuming of noncarpeted areas, which was done two times at a prescribed rate of 1 m^2/min, was followed by wet wiping with a detergent. They replaced one to three carpets and two items of upholstered furniture per housing unit. Their previous studies had shown that carpets could not be cleaned effectively with vacuuming alone. Where carpets could not be replaced, these were vacuum cleaned three times at a rate of 1 m^2/min, recognizing the limitations of this method.

The Cincinnati study recruited 307 children, including 16 children born to participating families during the study and 50 children from families recruited after the beginning of the study. In their main data analysis, the Cincinnati group excluded those children who were recruited after the start of the study, plus 31 children who were living in nonrehabilitated

housing suspected of having lead-based paint and four children (in two families) who had become lead-poisoned from other causes. Thus, data for 206 children were analyzed in the Cincinnati report.

The Cincinnati study abated soil on 140 parcels of land scattered throughout the neighborhoods. In CIN SEI, where soil abatement was performed in the first year, the arithmetic mean concentration dropped from 680 μg/g down to 134 μg/g. In the two groups where soil abatement occurred in the second year, CIN I-SE(D) and CIN I-SE(F), the soil lead concentration dropped from 262 to 125 μg/g and 724 to 233 μg/g, respectively.

If soil were the only source of lead in the neighborhoods, exterior and interior dust should have responded to the reduction in soil lead concentrations. Exterior dust lead loading decreased only slightly following soil and dust abatement, but returned to preabatement levels within one year. Exterior dust should provide a measure of exposure intermediate between soil and house dust. Where soil was abated, then exterior dust abatement should increase the rate at which the impact of this soil abatement can be observed on the interior dust of homes. But soil is not the only source of exterior lead, especially if the distance between the soil and the living unit entry way is more than a few hundred feet. In this case, the recontamination of exterior dust from sources other than soil complicates the interpretation of the movement of soil lead into the home or to exterior play areas.

Household dust was abated in the Boston and Cincinnati studies, but not in Baltimore. The BOS SPI and CIN SEI groups received interior dust abatement at the same time as soil abatement, the BOS PI-S received interior dust abatement in the first year, with soil abatement in the second year, and the CIN I-SE received interior dust abatement in the first year followed by soil and exterior dust abatement in the second year.

4.2 DESCRIPTION OF THE DATA

This section focuses on the actual data that formed the basis for the conclusions reached by the individual study reports. These data consist of measurements of soil, exterior dust (sometimes referred to as street dust), interior dust (house dust), hand dust, blood lead, exterior paint, interior paint, and drinking water. The age of the child and the date of collection were also included in some analyses. Tables 4-2, 4-3, and 4-4 summarize key

TABLE 4-2. SUMMARY OF BOSTON STUDY DATA[1]

	Round 1	Round 2	Round 3	Round 4
Median Soil Pb Conc. (μg/g)				
BOS SPI	2,396	125	115	193
BOS PI-S	2,307	-	2,084	278
BOS P-S	2,275	-	2,212	220
Median Floor Dust Pb Conc. (μg/g)				
BOS SPI	2,100	845	760	726
BOS PI-S	2,240	1,150	1,030	806
BOS P-S	2,200	950	1,300	862
Median Floor Dust Load (mg/m^2)				
BOS SPI	24	23	15	31
BOS PI-S	24	26	17	31
BOS P-S	40	28	19	37
Median Floor Dust Pb Load ($\mu g/m^2$)				
BOS SPI	52	23	16	24
BOS PI-S	59	27	18	28
BOS P-S	75	27	21	37
Median Window Dust Pb Conc. (μg/g)				
BOS SPI	13,240	11,217	21,125	8,780
BOS PI-S	19,667	10,000	15,650	6,870
BOS P-S	17,400	15,500	12,667	12,350
Median Window Dust Load (mg/m^2)				
BOS SPI	293	474	373	919
BOS PI-S	304	380	570	500
BOS P-S	239	239	504	797
Median Window Dust Pb Load ($\mu g/m^2$)				
BOS SPI	7,005	4,728	5,735	5,402
BOS PI-S	7,196	4,624	5,697	2,553
BOS P-S	4,179	4,441	5,559	6,018
Median Hand Pb Load (μg/pair)				
BOS SPI	6.75	4.0	3.5	12.5
BOS PI-S	6.75	5.5	2.0	7.15
BOS P-S	5.75	3.5	4.5	9.2
Median Blood Pb Conc. (μg/dL)				
BOS SPI	13	10	10	10
BOS PI-S	12	8	11	8
BOS P-S	12	9	11.5	10
GM Blood Pb Conc. (μg/dL)				
BOS SPI	12.36	9.11	9.90	9.07
BOS PI-S	11.70	8.01	10.74	7.11
BOS P-S	11.49	9.19	10.75	8.85

[1]Group assignments are as used in the Boston study report, and are the same as used in this report.

TABLE 4-3. SUMMARY OF BALTIMORE STUDY DATA[1]

	Round 1	Round 2	Round 3	Round 4	Round 5	Round 6
Median Soil Pb Conc. (μg/g)						
BAL SP	440	-	-	22	-	-
BAL P	409	-	-	-	-	-
Median Floor Dust Pb Conc (μg/g)						
BAL SP	1,600	-	-	1,068	-	-
BAL P	1,850	-	-	1,150	-	-
Median Floor Dust Load (mg/m^2)						
BAL SP	40	-	-	37	-	-
BAL P	37	-	-	38	-	-
Median Floor Dust Lead Load (μg/m^2)						
BAL SP	73	-	-	38	-	-
BAL P	72	-	-	41	-	-
Median Hand Pb Load (μg/pair)						
BAL SP	10.7	12.9	7.4	8.5	12.6	14.9
BAL P	13.6	14.8	9.5	6.0	17.3	13.0
Median Blood Pb Conc. (μg/dL)						
BAL SP	12.4	11.0	9.8	8.8	9.9	10.4
BAL P	10.6	10.2	9.2	7.4	8.0	8.0
GM Blood Pb Conc. (μg/dL)						
BAL SP	11.0	9.9	9.7	8.6	9.6	9.7
BAL P	10.9	10.5	9.1	7.8	8.1	8.4

[1]Group assignments are as used in the Baltimore study report, and differ from group assignments used in this report.

data for all three studies. For the most part, these data are the bases for the results and conclusions presented in the individual city reports, and also for the statistical analyses in Chapter 5 of this integrated assessment.

Each study produced similar information about the occurrence of lead in the environment. The data sets among the studies are not perfectly comparable, however, in that they differed in the timing of the collection relative to intervention (see Figure 2-1), the

TABLE 4-4. SUMMARY OF CINCINNATI STUDY DATA[1]

	Round 1	Round 2	Round 3	Round 4	Round 5	Round 6	Round 7
Median Soil Pb Conc. ($\mu g/g$)							
CIN SEI	680	134	142	103	122	166	132
CIN I-SE	237	247	240	262	125	182	138
CIN NT	339	346	330	256	331	267	266
Median Street Dust Pb Conc. ($\mu g/g$)							
CIN SEI	3,937	3,398	2,118	2,559	3,231		
CIN I-SE	3,665	3,416	3,411	2,275	3,040		
CIN NT	1,583	1,156	891	968	1,086		
Median Street Dust Load (mg/m^2)							
CIN SEI	454	242	363	452	310		
CIN I-SE	649	561	326	420	126		
CIN NT	624	755	481	477	654		
Median Street Dust Pb Load ($\mu g/m^2$)							
CIN SEI	1,162	789	641	968	808		
CIN I-SE	2,364	1,618	1127	943	371		
CIN NT	1,005	957	498	587	442		
Median Floor Dust Pb Conc. ($\mu g/g$)							
CIN SEI	362	346	325	474		158	
CIN I-SE	395	388	408	431		163	
CIN NT	229	224	209	213		162	
Median Floor Dust Load (mg/m^2)							
CIN SEI	418	134	135	197			
CIN I-SE	167	38	117	392			
CIN NT	147	126	161	200			
Median Floor Dust Pb Load ($\mu g/m^2$)							
CIN SEI	158	76	54	130	76		
CIN I-SE	69	18	58	243	108		
CIN NT	35	32	32	34	92		
Median Window Dust Pb Conc. ($\mu g/g$)							
CIN SEI	1,509	1,287	922	1,920	502		
CIN I-SE	2,000	1,572	1,306	2,017	592		
CIN NT	983	816	548	1,399	302		
Median Window Dust Load (mg/m^2)							
CIN SEI	710	433	254	4,524	966		
CIN I-SE	1,258	380	269	9,860	615		
CIN NT	2,170	2,534	324	8,573	648		
Median Window Dust Pb Load ($\mu g/m^2$)							
CIN SEI	983	426	242	15,385	397		
CIN I-SE	2,548	360	286	26,364	358		
CIN NT	1,782	1,111	172	12,849	227		

TABLE 4-4 (cont'd). SUMMARY OF CINCINNATI STUDY DATA[1]

	Round 1	Round 2	Round 3	Round 4	Round 5	Round 6	Round 7
Median Mat Dust Pb Conc. (μg/g)							
CIN SEI	109	738	549	767	659	-	-
CIN I-SE	132	939	702	722	889	-	-
CIN NT	100	373	349	405	332	-	-
Median Mat Dust Load Incremental Increase Per Day (mg/m^2/day)							
CIN SEI	-	6.5	7.7	4.4	28.2	-	-
CIN I-SE	-	18.7	4.7	4.9	16.6	-	-
CIN NT	-	1.8	2.0	2.7	12.2	-	-
Median Mat Dust Pb Load Incremental Increase Per Day (μg/m^2/day)							
CIN SEI	-	6.54	7.62	2.38	9.80	-	-
CIN I-SE	-	7.65	5.14	3.20	8.02	-	-
CIN NT	-	3.30	4.67	0.99	5.29	-	-
Median Entry Dust Pb Conc. (μg/g)							
CIN SEI	334	606	433	491	211	382	488
CIN I-SE	425	492	468	632	102	598	615
CIN NT	290	367	317	286	84	317	284
Median Entry Dust Load (mg/m^2)							
CIN SEI	386	113	230	590	12,671	97	301
CIN I-SE	272	70	142	1,394	17,889	161	513
CIN NT	348	238	294	373	14,509	148	1,080
Median Entry Dust Pb Load (μg/m^2)							
CIN SEI	112	104	167	250	2,502	56	150
CIN I-SE	95	38	70	588	2,700	103	302
CIN NT	157	80	88	106	1,714	58	264
Median Hand Pb Load (μg/pair)							
CIN SEI	6.0	5.0	5.0	12.0	12.5	-	-
CIN I-SE	7.0	7.0	5.0	10.0	8.0	-	-
CIN NT	3.0	4.0	3.0	5.5	7.0	-	-
Median Blood Pb Conc. (μg/dL)							
CIN SEI	9.2	-	7.0	8.0	-	7.9	8.3
CIN I-SE	10.8	-	9.2	8.9	-	8.0	8.8
CIN NT	9.0	-	5.9	6.8	-	6.4	7.8
GM Blood Pb Conc. (μg/dL)							
CIN SEI	8.8	-	6.9	8.8	-	8.2	8.7
CIN I-SE	10.8	-	9.3	8.6	-	7.6	8.9
CIN NT	8.3	-	5.7	6.8	-	7.2	7.8

[1]Group assignments are as used in the Cincinnati study report and differ from group assignments in this report.

spatial distribution of the sampling points relative to the expected exposure to the child, and the manner in which the data were reduced to a central tendency.

Data were collected in rounds. That is, during a specific period of time, samples were taken of soil, dust, etc., for a specific objective, such as establishing the concentration of lead prior to intervention. Usually a round lasted for several weeks, perhaps three to four months. It may be important to know when a sample was taken during a round, especially following intervention, in order to evaluate the impact on exposure. Consider the pathway from soil ➡ street dust ➡ house dust ➡ hand lead ➡ blood lead. One would expect, if soil alone (not house dust) were abated and the exposure were mainly through house dust, there would be a lag in time between abatement and response, and the impact of intervention might become greater with increasing time. Conversely, the impact of intervention might be reduced with time if there were recontamination, as would be expected if house dust were abated but soil or other sources were not.

It is important to know how well the soil concentration measurements and house dust concentration measurements actually represent the hypothesized pathway between soil and house dust. If the pathways are valid, it is possible to construct a simple exposure scenario for the individual child and to analyze these scenarios by structural equation modeling. For example, a young child may spend most of the time indoors, whereupon the exposure scenario becomes the lead that is available to the child through food, drinking water, air, and dust (see Figure 2-1). Each of these proximal sources of lead is influenced by one or more other sources of lead more remote from the immediate exposure of the child.

Some data are specific to the individual child, such as blood lead and hand lead. Some are specific for the living unit or family, and some are specific for the property. This distinction is important where there are several siblings in a family or several families in a dwelling. In such cases, a single numerical value for soil such as a mean or median for the premises could be heavily weighted if there were, for example, five children living on the same property.

4.2.1 Measures of Central Tendency for Property Level Soil and Dust

For soil and dust, there is a need to reduce multiple measurements within a round to a single representative data point, or central tendency, for each property or living unit. In

order to determine the appropriate central tendency for this measurement, the Scientific Coordinating Committee discussed several alternatives at great length without reaching a consensus. Therefore, different measures of central tendency were reported in each of the three studies. The following is an extended discussion of each of these measures, followed by an argument for the use of the arithmetic mean as the best measure in these circumstances.

The procedures for selecting a representative soil sample were based on the statistical distribution of data in each study. The Boston study used the median, giving no weight to extreme values. The Cincinnati study used the geometric mean, a method that is often used when the measured values are lognormally distributed, because it gives lesser weight to extreme values. The geometric mean is always lower than the arithmetic mean for any set of positive values and therefore may be an underestimate of the exposure to the child.

The distribution problem was approached differently in Baltimore, where the tri-mean was calculated as the weighted average of the first, second, and third quartiles:

$$X = \frac{Q_1 + 2Q_2 + Q_3}{4}, \qquad (4\text{-}1)$$

where

X = tri-mean, and

Q_n = nth quartile (Q_2 = median).

The tri-mean approach gives some consideration to the uneven distribution of values without unduly weighting the extremes. The tri-mean is equivalent to the arithmetic mean if the distribution is perfectly symmetric.

All three approaches assume that the sampling pattern is random and that exposure to soil is spatially random. Neither condition is strictly true in all three studies. One-third to one-half of the soil samples were taken 1 m from the foundation of the home, where concentrations are known to be higher than elsewhere. Because of playtime interests, parental instructions, or other influences, the child tends to play in specific areas that may represent less than 25% of the total soil area. All three studies collected some soil samples from play areas, where these could be identified.

It would seem reasonable that the ideal method for selecting a representative value should focus on the relationship between the soil and the child. The best measurement of central tendency is one that perfectly represents exposure to the child. This means that outside play activity patterns and exterior dust traffic patterns into the home must both be evaluated. In the case of outside play activities, a sample would be taken at each location where the child played and this sample would be weighted according to factors such as the time spent playing there and the frequency of hand-to-mouth activity during that time. Because this information is not available, a simplifying assumption is that weight should be given to the location of the sample rather than concentration. Location, not lead concentration, is the child's basis of choice for a play environment. An exposure weighted mean of the soil samples would seem to be the most direct approach. This would be an arithmetic mean of soil values corrected for the degree of exposure to the child. For example, a sample taken from bare soil in an area observed to be a play area would be given a high weighting factor for exposure. Grass covered areas with limited accessibility would be weighted on the low end of exposure. Although cumbersome, this method is feasible because such information was collected at the time of sampling in each study. The drawback is that the method emphasizes the direct, outdoor playtime contact between the child and the exterior dust, and does not consider other routes of dust exposure, such as soil ➡ household dust.

An alternative solution is to consider that the child has equal exposure to the entire surface of the soil. In this case, the perfect sample would be to scrape up this upper 2 cm of soil, homogenize it and take a sample. Theoretically, this is equivalent to sampling in a random pattern and taking the arithmetic mean of these samples. In this project, random locations were taken along lines specifically selected to represent the expected high- and low-concentration areas of the plot of soil. In this sense, the arithmetic mean is the best measure of the central tendency of soil data for a property, and is the statistic used in this report. Then, for *populations* of children at the neighborhood or higher level, the median or geometric mean of the arithmetic property mean is the preferred measure of central tendency for groups of children where extreme values should be suppressed.

4.2.2 Adjustments and Corrections to the Data

4.2.2.1 Subjects Dropped from Study

During the analysis of their data, the Boston group discovered that two children of the same family had apparently become exposed to lead-based paint abatement debris while staying at a house outside their neighborhood during a time when it was being remodeled. Both siblings had blood lead concentrations that had tripled in less than five months, between Rounds 1 and 3, from 10 to 35 μg/dL and 17 to 43 μg/dL. The Boston group analyzed their data with and without these children, eventually excluding these data from the analyses used to test their hypothesis. This Integrated Report accepts the conclusion that the data are outliers and also dropped them from further analysis.

There were four children identified by the Cincinnati investigators who were either chelated prior to or during the initial stage of the study, or who were victims of careless remodeling work. These four children were excluded from the Cincinnati analyses and from this assessment as well. Baltimore did not exclude any children based on medical intervention or careless remodeling.

The exclusion of these children in Boston and Cincinnati from statistical analyses was not arbitrary but followed extensive discussions among all participants in the project. This exclusion differs from the internal exclusion that occurs within specific statistical tests where several individuals may not meet the conditions of the test. For example, in one of their analyses, Baltimore compared the blood lead concentrations for children in Rounds 1 and 6. By specifying that a child must have been present for both rounds, this selection excluded children recruited in Rounds 2 and 3, and any children whose blood was not sampled in Round 6. For other statistical analyses, some or all of these children may have been included. For all statistical analyses in this report that involve blood lead measurements for specific rounds, a child is included if a blood lead measurement was taken for that round and if other data required for the analysis are also available.

4.2.2.2 Unit Conversion

All data were converted to common units, usually metric. Corrections were made for analytical blanks or similar analytical adjustments, as reported by each individual city

research team. In this assessment, all data for soil and dust were adjusted by the interlaboratory correction factor specific for each study and shown in Table 3-6.

4.3 STUDY DESIGNS

4.3.1 Design Differences

Table 4-5 describes the design differences among the three studies. While considerable effort was made to coordinate the study designs so as to assure the highest possible degree of comparability among study results, the investigators in the three cities faced different design issues that precluded carrying out completely identical or equivalent studies. Thus, although participant recruitment and certain other aspects were similar across the three cities, some salient differences are also worth noting.

The first difference was that there were different levels of remediation or treatment among the cities. Boston used two comparison or reference groups in addition to the soil abatement group, whereas Baltimore used only one such group. In the Cincinnati study, there were three levels of intervention. Also, the trigger level for soil lead removal varied somewhat across the cities. In the Baltimore and Cincinnati, a maximum level of 500 μg/g or greater in the parcel or residential property triggered soil removal. In contrast, all Boston properties had mean or median soil concentrations exceeding 1500 μg/g. Properties recruited in the Boston study were scattered across four large neighborhoods or urban areas, although households were assigned at random to the treatment group for soil removal and not specifically limited to any given neighborhood. This randomization approach in Boston provides a more thorough statistical treatment of multiple sources of lead and analysis of environmental cofactors. The Baltimore study was carried out in two large neighborhoods, with soil lead removal restricted to only one of the neighborhoods (Lower Park Heights). Most houses above the soil lead trigger level in the Lower Park Heights neigborhood in the Baltimore study had yard soil removed, but some did not, and no house in Walbrook junction had soil removed. The Cincinnati study was carried out in six smaller neighborhoods, with soil and exterior dust removal only carried in the Pendleton neighborhood. In the Cincinnati study, all parcels in Pendleton above the soil lead trigger level had soil removed in the first

TABLE 4-5. DESIGN DIFFERENCES AMONG THE THREE STUDIES

Design Feature		Boston	Baltimore	Cincinnati
Number of treatment groups		3	2	3
Number of rounds with blood Pb measurement		4	6	5
Interval between abatement and final blood Pb measurement (months)		22	10	20
Soil removal trigger level ($\mu g/g$)		1,000	500	500
Paint stabilization		Interior	Exterior	None[a]
Number of neighborhoods		4	2	6
Participant recruitment		Volunteer	Volunteer	Volunteer
Treatment assignment to participants		Random	By Neighborhood	By Neighborhood
Control groups with no intervention		No	No	Yes
Age structure of participants (%)	0-1	2.7	8.6	29.9
	1-2	24.0	17.6	17.2
	2-3	34.0	18.1	17.6
	3-4	34.7	18.4	15.8
	4-5	4.7	20.3	14.0
	5-6		14.5	5.4
	6→		2.5	
Ethnicity (%)				
Black		51	100	97
Hispanic		15	0	0
White		7	0	2
Other		27	0	1
Male/female ratio		47/53	48/52	44/56
Blood sample collection	R1	1-2 mo preabate	24 mo preabate	1-2 mo preabate
	R2	3-4 mo after R1	12 mo preabate	
	R3	10 mo after R1	5-8 mo preabate	3-4 mo after R1
	R4		8-10 mo after R3	11 mo after R1
	R5	22 mo after R1	14-16 mo after R3	
	R6		18-20 mo after R3	16-18 mo after R1
	R7			22-24 mo after R1

[a]Dwelling units had been thoroughly rehabilitated 20 years prior to study, leaving little exposed lead-based paint.

year. Soil abatement occurred during the second year in the Back, Dandridge and Findlay neighborhoods, and in the control groups at the end of the study.

Paint was stabilized inside all Boston houses and outside all Baltimore houses, but not in Cincinnati where it was believed that only gut-rehab houses had been recruited into the study. No Baltimore residence received interior abatement, either of dust or lead paint, whereas the majority of the residences in the Boston and Cincinnati studies received interior dust abatement whether or not they were in the soil removal treatment group. Exterior dust abatement was performed only in Cincinnati.

Demographic differences among study populations should also be noted. The age distribution of children at the time of abatement differed among the three studies. The Baltimore group had more children of age at least four years, since many of the children had been initially recruited up to 2 years earlier. Almost all of the children initially recruited in the Baltimore study were of African-American ancestry; by the final phase of the study, 100 percent of the study group was African-American. The Cincinnati study group was slightly more diverse, with a small percentage of Caucasians of Appalachian origin. The Boston group was the most diverse, with substantial subgroups of white and Cape Verdean children, and also with a large percentage of African-American children. Percentages of male and female children differed somewhat among the cities. While all of these inner city households tended to be economically disadvantaged, the majority of the households in Baltimore were occupied by the property owner, which was uncommon in the other two cities.

Lastly, as for biological measurements indexing changes in lead exposure, each study involved collection of preabatement and postabatement blood samples and their analyses. However, the numbers of sampling points varied across the studies. The studies had four to six rounds of blood lead collection, with one to three pre-abatement rounds, a short-term post-abatement round (about two or three months), and two to three rounds up to two years post-abatement.

4.3.2 Strengths and Weaknesses of Study Designs

In an ideal situation, each study would have been designed around a neighborhood where soil was a significant source of dust in the child's environment and this soil contained an amount of lead sufficient to impact the child's exposure. There would also be no other sources of lead in the child's environment, and the child's history of lead exposure would

have been stable. The study would incorporate all children in the neighborhood and these children would be demographically similar to a representative sample of children across the United States. Their behavior and activity patterns would also be similar and representative. Children would be randomly assigned to a study group, and the population would be sufficiently large to test the main hypothesis as well as any other question that might arise concerning sibling, ethnic, age, and sex effects. The sample design plan should be sufficient to establish the pattern of lead exposure for the child population prior to intervention, including seasonal cycles and long term trends in blood lead and dust lead loading.

None of the three studies in this project met these ideal conditions, nor could any other neighborhood in any city. The issue then is whether any departure from this ideal design seriously impacted the conclusions that the study could have made under ideal circumstances or did make under these more realistic circumstances. In this respect, the strengths and weaknesses of the three study designs are discussed and the hypotheses of the individual studies are reevaluated.

The strong points of the Boston study are that it was designed as a group of demographically similar neighborhoods where soil was a significant source of dust and lead exposure. It appears that the children were also similar in terms of behavior and activity patterns and diverse in age, ethnicity, and sex. The main weaknesses in the Boston study are that some children were excluded from selection into the study because of high or low blood lead concentrations. This truncation of blood leads above 24 μg/dL, excluding these children may have substantially diminished the impact of intervention, on the assumption that children with higher blood lead concentrations would show a greater response to reduced exposure. This hypothesis is tested in this assessment.

There was also a sufficient amount of lead from interior and exterior lead-based paint in most residences to partially obscure the impact of soil abatement. The Boston properties were not contiguous, so that no measure of neighborhood level lead exposure of intervention can be made. Even though the Boston study may not represent typical U.S. urban neighborhoods, the study is likely applicable to a broad range of circumstances because the experimental treatment was assigned at random to children living on properties in four distinct neighborhoods, and this randomization in study design is likely to have eliminated many neighborhood level confounding factors.

The Baltimore study design incorporated two demographically similar neighborhoods, one designated as a treatment group and the other a control. Soil may have been a significant source of dust in the child's environment, but the soil and dust sampling protocols were inadequate to test this hypothesis. The Baltimore group identified up to four parcels of soil on each property and abated any parcel where the maximum soil concentration exceeded 500 μg/g. In those cases where the abated parcel, by circumstance, was not one that the child would play on or one that would contribute in any other way the child's lead exposure, then there would be no change in the child's lead exposure in response to abatement and no expected decrease in blood lead, even though the property was in the "abated" group. This type of error, normally called "misclassification", could not be evaluated in this assessment because the soil samples were not identified by parcel identifiers.

For blood lead and hand lead, the Baltimore study sampled three preabatement and three postabatement rounds. While it is possible that these data may be sufficient to identify a seasonal cycle or a long term trend similar to that discussed in Section 2.3.1, analyses for these effects were not made by either the Baltimore report or in this assessment. In the Baltimore study there was insufficient lead in the soil to demonstrate an effect of abatement and there was a substantial amount of lead in exterior and interior paint to obscure the impact of intervention. Windows were not included in the sampling pattern for house dust. The floor dust was sampled only once for most children, which is not frequently enough to detect changes in the child's exposure to environmental lead. For some children there was a postabatement dust sample taken.

The Cincinnati study alone evaluated intervention on a neighborhood wide basis. The frequency of environmental sampling was sufficient to identify important features of environmental dust mobility. However, soil appears not to have been a major contributor to house dust, nor was the soil lead concentration sufficiently high to impact exposure to environmental lead through exterior dust. Most of the lead in exterior dust appears to have come from nonsoil sources. Of the six neighborhoods in the Cincinnati study, one (Back Street) was too small to continue in the study, and another (Glencoe) may have been demographically distinct from the rest. Children were assigned to study groups based on their resident neighborhood rather that randomly assigned from the group of neighborhoods,

and these neighborhoods were grouped by the Cincinnati investigators into treatment and control groups in a non-random fashion.

4.3.3 Modifications of the Hypotheses

Each study developed a specific hypothesis that was intended to be tested by the data and observations from the original study design. For the purposes of clarification only, this report restates the original hypotheses in a form that is more amenable to statistical interpretation. This clarification will aid the reader in understanding the statistical analyses that are presented in Chapter 5. In the case of Boston and Cincinnati, the original hypotheses are broken into three subhypotheses.

The first subhypothesis for Boston identifies their group "S" as the primary treatment group, and the second hypothesis identifies group "A" as a positive control group. An analogous distinction is made for the Cincinnati study, with the first subhypothesis for the soil abatement neighborhood of Pendleton, and the second subhypothesis for dust abatement neighborhoods Back Street, Dandridge, and Findlay. No subhypotheses were designed for the Baltimore study, since the neighborhood effects were confounded with treatment effects. In the EPA reanalyses, additional subhypotheses were developed based on *post hoc* differences between certain subgroups. The formal statement of the original Boston hypothesis is:

> *A significant reduction (equal to or greater than 1,000 μg/g) of lead in soil accessible to children will result in a mean decrease of at least 3 μg/dL in the blood lead levels of children living in areas with multiple possible sources of lead exposure and a high incidence of lead poisoning.*

The actual hypothesis that was tested is similar to this and might be restated as follows, in the null form:

> *(i) A one-time reduction of at least 1000 ppm in average soil lead concentration of residential property without substantial deteriorating exterior lead paint, accompanied by remediation of interior household dust and control of recontamination of interior dust by stabilization of interior paint, will not result in a reduction of blood lead in children living at the residence.*
>
> *(ii) A one-time dust lead abatement inside a residential property without substantial deteriorating exterior lead paint, accompanied by control of*

recontamination of interior dust by stabilization of interior paint, will not result in a reduction of blood lead in children living at the residence.

(iii) The reduction in (i) will not be greater than the reduction in (ii), if any.

The original primary Cincinnati hypothesis, pertaining to blood lead levels, was stated as

(1) A reduction of lead in residential soil accessible to children will result in a decrease in their blood lead levels.

(2) Interior dust abatement, when carried out in conjunction with exterior dust and soil abatement, would result in a greater reduction in blood lead than would be obtained with interior dust abatement alone, or exterior dust and soil abatement alone.

To reflect actual experimental conditions, this hypothesis could be modified as follows and restated in the null form

(i) A one-time reduction of lead in accessible soil and in street dust in a neighborhood, accompanied by abatement of household dust in the child's apartment or residence unit, will not result in a reduction of blood lead in children living in gut-rehab housing in the neighborhood.

(ii) A one-time reduction of lead in household dust in the child's apartment or residence unit, will not result in a reduction of blood lead in children living in gut-rehab housing in the neighborhood.

(iii) The reduction in (i) will not be greater than the reduction in (ii), if any.

The original Baltimore hypothesis, stated in the null form, is

A significant reduction of lead ($\geq$ 1,000 μg/g) in residential soil accessible to children will not result in a significant decrease (3 to 6 μg/dL) in their blood lead levels.

A restatement of this hypothesis that takes into consideration the actual preabatement conditions and stated in the null form is

A one-time reduction of at least 500 ppm in the maximum lead concentration in yard soil, even when not accompanied by abatement of household dust or lead paint inside the child's apartment or residence unit, will not result in a reduction of blood lead in children living in housing in which exterior lead paint has been stabilized.

The actual statistical hypotheses and subhypotheses are all expressed as "null hypotheses", that abatement has no effect, makes no difference, or causes no change compared to no abatement, after other processes or changes have been taken into account. This differs some what from the original statement of the hypotheses by the investigators. To clarify this distinction, a little algebraic notation may be helpful. Let "E" denote the size of the abatement effect, for example, a reduction in blood lead concentration in the abatement group relative to a control group, and let s.e.(E) denote the estimated standard error of the effect size estimate. Most statistical tests carried out by the investigators and by EPA involve use of statistics that are essentially equivalent to a "t" statistic, defined by

$$t = \frac{E}{s.e.(E)}$$

For the original hypothesis proposed by the Boston investigators, where a blood lead reduction of at least 3 μg/dL, was stipulated, a somewhat different t statistic would be appropriate,

$$t' = \frac{(E-3)}{s.e.(E)}$$

Similar alternative hypotheses were adopted in a roughly similar form by the Baltimore and Cincinnati investigators for the purpose of power calculations to aid in study design. In this report as well as in their reports, inferences were in fact based on statistics like "t" for testing no effect.

4.4 INDIVIDUAL STUDY CONCLUSIONS

In their report following the first phase of their study, the Boston group stated their conclusions:

> *"...this intervention study suggests that an average 1,856 ppm reduction in soil lead levels results in a 0.8-1.6 μg/dL reduction in the blood lead levels of urban children with multiple potential sources of exposure to lead."*

Following the second phase of the study, they concluded (Aschengrau et al., 1994):

"The combined results from both phases suggest that a soil lead reduction of 2,060 ppm[1] is associated with a 2.2 to 2.70 μg/dL decline in blood lead levels."

The basis for their initial conclusions consisted of an analysis of variance comparing mean blood lead changes among the three intervention groups, paired t-tests for within group effects, and analysis of covariance with one-at-a-time adjustment for age, SES, race, sex, paint, water, and mouthing behavior. The analysis of covariance was performed using no transformation of blood lead data, which appeared to be normally distributed.

The conclusions from the second phase of the study are based on additional analyses of phase one and phase two data using two-way analysis of variance (ANOVA) with repeated measures. Soil was abated for the two original control groups (BOS PI-S and BOS P-S) at the beginning of phase 2. The reduction in blood lead is based on pre- and postabatement measurements of all three groups.

The Baltimore group stated their conclusions as follows:

"Statistical analysis of the data from the Baltimore Lead in Soil Project provides no evidence that the soil abatement has a direct impact on the blood lead level of children in the study."

"In the presence of lead-based paint in the children's homes, abatement of soil lead alone provides no direct impact on the blood lead levels of children."

The basis for these statements consisted of an adjusted and unadjusted analysis of selected covariates. The natural log of the blood lead of children in the treatment group showed no significant difference from the natural log of the blood lead of children in the control group, even when adjustments were made for: age, SES, hand lead, season, dust, soil, sex, weak mouthing behavior, or strong mouthing behavior. These analyses were made on two sets of data. The first set consisted of all children enrolled in rounds one and six. The second group consisted only of children enrolled in all six rounds.

The Cincinnati conclusions can be paraphrased as follows based on their individual report:

Following interior and exterior dust and soil lead abatement, blood lead concentrations decreased (in Area A) from 8.9 to 7.0 (21%) but increased to 8.7,

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[1] This value for soil, 2,060 ppm, cited in their published report, was not adjusted by the Boston group with the interlaboratory correction factor of 1.037 in Table 3-6.

10 months postabatement. Following interior dust abatement alone blood lead concentrations decreased from 10.6 to 9.2 (13%) four months postabatement and were 18% below preabatement 10 months postabatement. With no abatement, blood lead levels decreased by 29 and 6% during these same time periods. Other comparisons also revealed no effects of the soil or dust abatement.

There was no evidence that blood lead levels were reduced by soil lead or dust abatement in Area A (with soil, exterior dust, interior dust abatement). There was a slight reduction (net reduction over control area) of 0.6 μg/dL in Area B that might be attributed to interior dust abatement. This difference is not statistically significant.

The basis for the Cincinnati conclusions was a comparison of environmental and blood lead data for the three treatment groups from Rounds 1, 3, 4, 6, and 7 and of additional environmental data from Rounds 2 and 5.

5. RESULTS OF INTEGRATED ANALYSES

5.1 CONCEPTUAL APPROACHES TO EVALUATING RESPONSE TO ABATEMENT

Many statistical procedures rely heavily on an analysis of the correlation structure of the data (how one variable changes in response to or in conjunction with another, covarying variable) within a single measurement period or round of measurement, and on the longitudinal structure of the sampling scheme, where several rounds of measurement are made. In preparation for a detailed description of statistical methods, the following discussion briefly reviews the variability of the key variables and the correlation structure among their covariates.

In designing the three individual studies, the investigators expected that blood lead, handwipes, and house dust would be predictors of childhood lead exposure. Changes in blood lead levels, in hand lead levels, and in household dust lead levels were expected to occur in response to effective intervention and in response to other biological and environmental changes that occurred independent of intervention. Many of these patterns of change were discussed in Chapter 2 and are reviewed briefly here. As shown earlier in Figure 2-7, blood lead concentrations in young children often increase up to ages 2 or 3 years, which are peak ages for ingestion of soil and dust during play, and then decrease slowly in older children (U.S. Environmental Protection Agency, 1986; Clark et al. 1988). Hand lead loadings increase steadily with age (Bornschein et al., 1988). House dust lead levels may increase or decrease as changes in sources or exposure pathways cause changes in the amounts of dust that move through the environment and in the amounts of lead in that dust.

Childhood blood lead concentrations are, to some extent, a measure of the recent history of lead exposure and may respond to environmental changes in lead within a time frame of a few months (see Figure 2-4). Reductions in blood lead due to reductions in exposure possibly attributable to intervention might be somewhat attenuated by the remobilization of lead in bone tissue. Figure 2-4 shows the complexity of biokinetic translocations of lead when the total body burden is decreasing. If the total lead exposure of

the child decreases, there seems to be no doubt that the blood lead concentrations would also decrease, but measurements of this decrease would be complicated by the remobilization of bone and other tissue lead, and interpretation of these measurements would be complicated by the knowledge that the reduction in exposure cannot be fully attributable to reductions in soil lead exposure.

Changes in blood lead must be interpreted in the context of four time-dependent effects that are independent of each other, as summarized below.

(1) Seasonal changes in children's blood lead concentrations have been reported in several longitudinal studies. These usually show a peak in blood concentration during the late summer months.

(2) Changes occur with age during early childhood such that blood lead concentrations usually peak between 18 and 27 months of age.

(3) Long-term changes in national baseline levels of exposure, believed to be mostly from reductions of lead in gasoline and in food, are reflected in a downward trend for U.S. childhood blood lead levels observed since 1978.

(4) Further changes can be attributed to household- and neighborhood-level interventions of the types reported in this project.

The first three of these effects were discussed in Section 2.3 and are summarized here. The fourth is the main topic of this chapter.

5.1.1 Expected Impact of Intervention

5.1.1.1 Expected Impact of Soil Abatement on Exterior and Interior Dust

The key to understanding the impact of soil abatement on interior dust is to observe changes in the three components of the interior dust measurement: lead concentration (micrograms of lead per gram of dust), lead loading (micrograms of lead per square meter), and dust loading (milligrams of dust per square meter). Where there was no interior dust abatement, the lead concentration in interior dust should decrease gradually over time in response to soil abatement, provided that the influence of other sources such as lead-based paint have been minimized. Also, the lead loading should decrease if the dust loading remains constant. If interior dust abatement has occurred, the lead concentration should gradually reach some new equilibrium concentration determined by the sources of lead and

dust, and the lead loading and dust loading should increase and decrease in tandem according to the pattern of (a) dust movement to the house or other play environment and (b) frequency and efficiency of housecleaning.

Chipping and peeling lead-based paint can contribute vast amounts of lead to household dust over a relatively short period of time. A single chip 5 cm x 5 cm at 6 mg Pb/cm^2 contains enough lead (150,000 μg) to increase the concentration of lead in a house with 7 g dust (54 mg/m^2 x 130 m^2) by 21,000 $\mu g/g$. In this project, chipping and peeling paint was removed by paint stabilization. But there is no measure of the amount of lead that passes from paint to housedust by other routes, such as abrasion, weathering, or microscopic flaking. In the interior of Boston homes, paint stabilization also included repainting the surfaces with a coat of latex primer paint to slow or minimize recontamination of interior house dust from paint.

The confounding effect of lead-based paint can be minimized in three ways: (1) exclude homes with lead-based paint; (2) stabilize the paint so that the rate of incorporation to house dust is minimized; and (3) compare measurements for areas where the influence of lead-based paint is probably high relative to soil with data for areas where the relative influence is low.

Exterior dust was measured and abated in the Cincinnati study only, and the results suggest a highly fluctuating rate of recontamination from non-soil sources. With a source of lead of this magnitude at the neighborhood level, it is virtually impossible to measure the impact of soil abatement on house dust directly. However, if abatement is considered on the broader scale, where neighborhood cleanup would include soil, external dust, and any other sources of lead external to the home, then the house dust measurements made immediately inside the homes can be used to assess this "total neighborhood abatement".

5.1.1.2 Expected Impact of Soil and Dust Abatement on Hand Lead Loading

It was expected that hand dust would serve as a surrogate measure of changes in exposure following abatement, to augment information about blood lead changes. Hand dust reflects the child's recent exposure (since the latest hand washing) but is only a measure of lead loading, not lead concentration or dust loading, because the total amount of dust is not measured. Consequently, it is not possible to infer the source of lead (soil or paint) by

differences in concentration, nor is it possible to assess the housekeeping effectiveness by observing changes in hand dust loading, as with house dust loading. In fact, even the changes in hand lead loading proved to be a poor indicator, in this project, of changes in blood lead.

5.1.1.3 Expected Impact of Soil and Dust Abatement on Blood Lead Concentrations

Soil lead remediation in residential yards was expected to have both direct and indirect effects on childhood lead exposure. The direct effect of removing lead contaminated soils is to prevent access to the lead in the soil. For most children, direct exposure to lead in soil is likely to come from fine particles of loose soil or exterior surface dust that adhere to the child's hands and are transferred to the child's face and mouth during hand-to-mouth contact that is part of normal behavior for preschool children and infants. Most children do not eat large quantities of soil. Quantitative estimates of soil ingestion by children are limited and highly variable, probably due to differences in methodology and choice of tracer element used in determining the estimate (U.S. Environmental Protection Agency, 1989).
By calculating a single estimate of soil ingestion for each subject, Stanek and Calabrese (1995) determined the median daily soil ingestion for 64 children living in Amherst, MA. Mean soil ingestion estimates were 45 mg/day or less for 50 % of the children and 208 mg/day or less for 95% of the children. Some children may regularly ingest a large amount of soil either in a non-pica situation where the child is on the upper tail of a unimodal distribution for soil ingestion, or in a pica situation, where the child habitually eats nonfood objects such as soil. Some adults (soil or clay eaters) are known to experience geophagia, but these are atypical conditions and are not appropriate for assessing soil risks for the majority of children.

Soil is also a source of lead in dust in the child's play areas. Soil in the residential yard may be tracked into the house by its occupants (including pets), and fine exterior dust particles may become re-entrained and carried into the house as micro-scale air contaminants. Fine dust particles may adhere to the child's hands and may contaminate food during its preparation. This dust is usually a more important medium of lead intake than is the direct ingestion of soil. The soil lead pathway is one of several possible sources of lead in dust (see Figure 2-3).

Blood lead concentrations should respond to soil and dust abatement through the impact of intervention on two routes of exposure: (1) hand-to-mouth activity, reflecting the impact of interior house dust and exterior play area dust on exposure; and (2) food contamination, reflecting the incorporation of house dust in food during kitchen preparation. There was no measure of the incorporation of house dust into food during this project. Intuitively, the impact of interior dust abatement should be the same, or at least comparable, for food and hand dust. In some homes, however, lead-based paint is found primarily in kitchens and bathrooms, where the remobilization of lead in dust from lead-based paint following stabilization would have a greater impact on food than hand dust. There is a limited amount of data, not yet analyzed, where kitchen floor dust can be compared to bedrooms and other living areas, and likewise for window wells. Most of these data, however, are from the Cincinnati study, where there was a minimum influence of lead-based paint.

5.1.2 Evaluation of Specific Statistical Approaches

The studies in each of the three cities are characterized as longitudinal intervention studies with fixed treatment groups, and there are at least seven statistical methods that could be used to analyze their data. Of the seven methods, ranging from the simplest analysis of blood lead data alone to complex analyses of changes in blood lead concentrations that occur in response to changes in environmental lead over time, this assessment used four methods for each of the three studies. In this introductory section, each of the seven methods is discussed, with the rationale given for whether or not the method would be appropriate for this assessment. A more detailed description of the statistical methodology is given in Sections 5.4, 5.5, and 5.6. The seven methods are

1. Cross-sectional analysis of variance
2. Cross-sectional analysis of covariance
3. Cross-sectional structural equation models[1]
4. Longitudinal analysis of covariance
5. Repeated measures analysis of variance[1]
6. Repeated measures analysis of covariance[1]
7. Longitudinal structural equation models[1]

[1]Methods used in this report.

By using more than one method, and by using methods that, in general, are more comprehensive in their treatment of the data, this assessment is able to increase the strength of the conclusions and, in some cases, provide a more detailed explanation of the nature of the effect than appeared in the individual city reports.

The statistical methods described above differ primarily in the complexity with which they treat the data. This complexity arises from the stepwise insertion of mathematical terms into what is otherwise a very simple equation, usually in a linear form. These equations are presented below and, as an aid to the reader, descriptive graphics and simple mathematical solutions to the equations are provided.

5.1.2.1 Cross-sectional Analysis of Variance

The first method, not used in this assessment, is a simple cross-sectional analysis of variance, with each round treated separately, as illustrated in Figure 5-1. In Figure 5-1, we show two groups of children, denoted "A" and "B". These groups are observed at several times (rounds), at least once before and after the soil abatement, shown as separated by a vertical dashed line. The amount of lead present in the residence and in the child at each round is suggested by the number of dots in each figure. The changes from soil abatement in group B are shown by the smaller number of dots in the yard and residence, and the expected effect of abatement is shown by the smaller number of dots in the child, implying a reduction in blood lead and therefore a reduction in the total body burden of lead. A simple analysis of variance would compare the group mean blood lead between Group A and Group B. Before abatement, the two groups appear roughly equal. After abatement, Group B has a lower blood lead than Group A. The effect size would be calculated as the difference between the groups after abatement. This approach is a simplified description of one of the analyses used in the Baltimore report. The children in each group may be somewhat different at each round, depending on attrition and recruitment. An alternative version of this approach uses the change in blood lead in each child from a preabatement round to a postabatement round, which is a simplified description of one of the approaches used in the Boston report.

The equations of this method are fairly simple and form the basis for the more complex analyses that use computing capabilities that are much stronger than were available earlier for

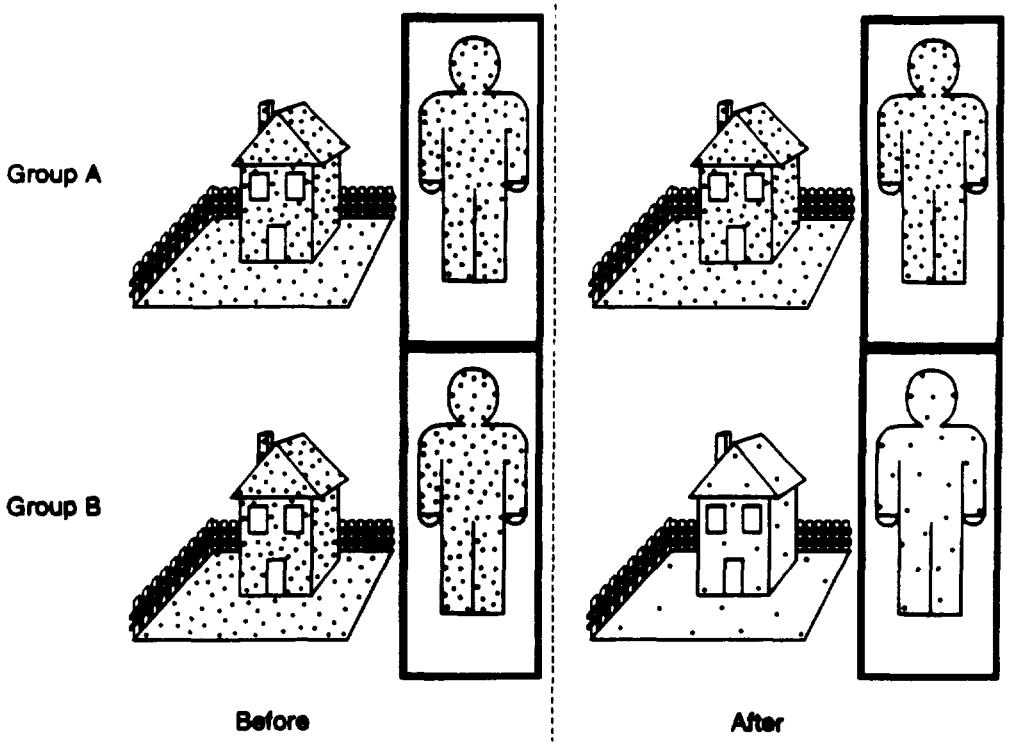

Figure 5-1. Analysis of variance. Blood lead is compared across treatment groups at each round. Environmental measurements of dust and soil are not part of the statistical analyses. The density of dots in the house and child figures shows a scenario in which soil abatement in group B reduces soil lead, house dust lead, and blood lead. Comparison of Group A and Group B is made only at the postabatement round, where the vertical dashed line separates pre- and postabatement scenarios.

routine analyses. For an analysis of variance of factors that influence blood lead concentrations, only the group effect is evaluated, which means that the statistical test simply looks for a systematic difference between groups, while excluding the effects of covariant data such as age and sex. If the children in each group are otherwise equivalent, then the group effect is, by inference, the effect of treatment on the child. In a representative equation for this method, the response variable, Y_{ir} (e.g. blood lead for child *i* at round *r)* is estimated by the equation

$$Y_{ir} = G_{gr} + e_{ir} \quad (5\text{-}1)$$

Y_{ir} is the response variable for child i at round r

G_{gr} is the fixed mean response for treatment group g at round r

e_{ir} is an error term, referred to as measurement error, for child i at round r

The term G_{gr} can be used to calculate the effect size. Each of these statistical methods allows estimation of an effect size, which cannot be defined as an intrinsic property of a group or treatment. Rather, it is defined in terms of a relationship to a control group or combination of control and treatment groups. It is important to compare treatment groups with control groups because even "control" groups may change over time or may respond to nontreatment environmental influences. The effect size, ΔG_g, for the analysis of variance method would be calculated as the difference in fixed effect between two treatment groups at each round separately:

$$\Delta G_g = G_{1r} - G_{2r} \quad (5\text{-}2)$$

In the case where some households in a group have multiple siblings, analysis of variance could also be used to separate the treatment group effect from a random household effect ($H_{h(g)}$) for each round r separately, using the equation:

$$Y_{ir} = G_{gr} + H_{h(g)} + e_{ir} \quad (5\text{-}3)$$

then estimating the treatment group effect at each round as above,

$$\Delta G_g = G_{1r} - G_{2r} \quad (5\text{-}4)$$

The advantage of analysis of variance is that this method can deal with multiple groups or treatments. The limitations are that it can be applied to only one response variable and to the before/after treatment differences for that variable. The method ignores the correlation structure among the measurements and, thus, does not have the capability to adjust for covariates.

5.1.2.2 Cross-sectional Analysis of Covariance

The six remaining methods achieve a progressively more refined estimate of the group effects, mainly by analysis of the correlation structure within the data set. Cross-sectional analysis of covariance, illustrated in Figure 5-2, is similar in form to cross-sectional analysis of variance, adding one or more terms that adjust the estimate for interaction of covariates. Figure 5-2 is the same as Figure 5-1, but the arrows suggest that child's blood lead used in the analyses is adjusted for soil lead and dust lead concentrations. The hypothesis used here is that there is a more or less strong relationship between dust lead and blood lead, and between soil lead and blood, as two separate media to which the child may be exposed. Additional adjustments may be made for other household-specific covariates such as socioeconomic status (SES) or race, or for child-specific covariates such as age, gender, or mouthing behavior. Adjustments for these covariates allows a better understanding of the possible processes or mechanisms of abatement, including separation of group mean differences into those components that related to changes or differences in soil lead and dust lead, and other effects that are not attributable to abatement changes in soil lead or dust lead. However, the analysis of covariance (called ANCOVA) in this formulation does not use the changes in covariate values before and after abatement. Some models in the Baltimore report used this approach. Similar models in the Cincinnati report used differences in blood lead or hand lead vs changes in environmental lead. This method makes adjustments for between-group effects and environmental covariance, but still doesn't estimate the before/after effect of intervention.

In this case, a term $X_{ir}B_{gr}$ is added to Equation 5-1 to account for the covariates, and each round is again analyzed separately:

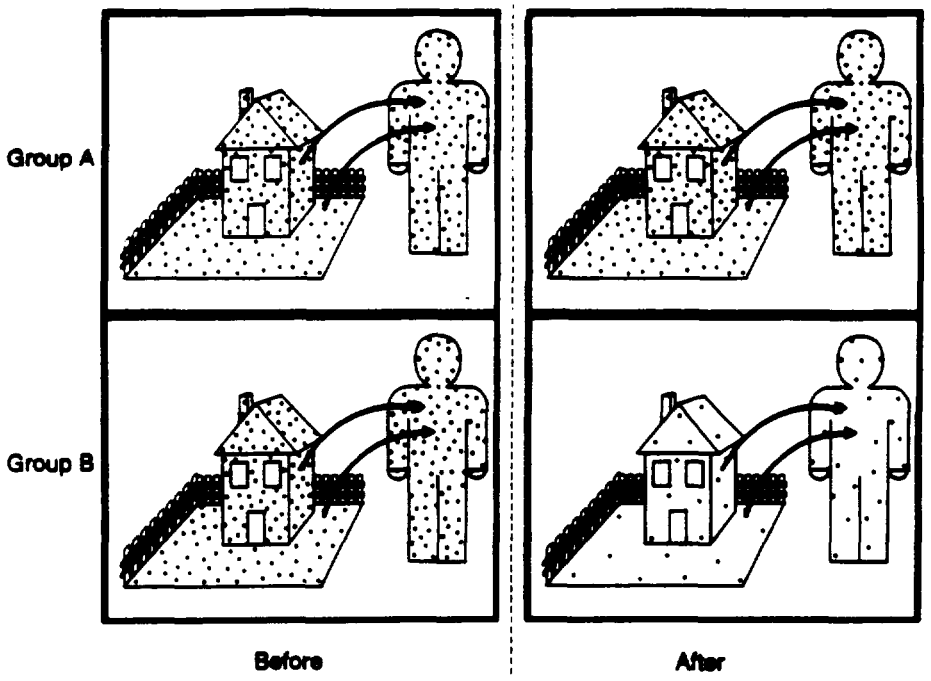

Figure 5-2. Analysis of covariance. Arrows show that the individual child blood lead concentrations are adjusted for soil lead and dust lead concentrations or loadings in the child's residence, and may also be adjusted for other household-specific or child-specific covariates such as age or gender. However, blood lead is compared across treatment groups at each round with no analyses of effects between rounds.

$$Y_{ir} = G_{gr} + X_{ir}B_{gr} + e_{ir} \qquad (5\text{-}5)$$

X_{ir} is the covariate value for child i at round r

B_{gr} is the covariate effect for group g at round r

Note that Equation 5-5 has taken on a linear form and, with multiple covariates, could be the basis for multiple linear regression analysis. The effect of treatment group after covariate adjustment would still be calculated as equation 5-4, but also has a component that is related to the covariate difference, which may be different in each group,

$$\Delta(XB) = X_1B_{1r} - X_2B_{2r} \qquad (5\text{-}6)$$

where X_1 is a typical covariate value for Group 1 and X_2 a typical value for Group 2. Like analysis of variance, this method can deal with multiple groups or treatments. Although it also ignores the correlation structure among the variables, the method can be used to adjust for covariates, and these covariates may be either numerical or categorical. A limitation of analysis of covariance is that covariates are assumed to be known without measurement error, and this becomes a problem when the analysis is of two environmental variables, each with its own undetermined measurement error. Neither cross-sectional analysis of variance nor cross-sectional analysis of covariance was used in this report or in the individual city reports.

5.1.2.3 Cross-sectional Structural Equation Models

Cross-sectional structural equation modeling (Figure 5-3) has the advantage of modeling the relationships between several environmental pathways simultaneously, such as soil to dust, dust to blood, soil to blood. In this simple form, the method provides no longitudinal information, and therefore cannot fully treat such time dependent variables as blood lead when the main contributor to blood lead at time t is the blood lead at time t-1 (see Figure 2-4). Figure 5-3 is essentially the same as Figure 5-2, with arrows showing the relationship of soil lead and house dust lead to blood lead. There is an additional arrow showing a relationship between house dust lead and soil lead. This allows assessment of the hypothesis that house dust lead has a component attributable to soil lead, and possibly other components that are not attributable to soil lead (such as lead-based paint or secondary occupational exposure). It then becomes possible to assess the total relationship of soil to blood lead, including the indirect soil-to-dust-to-blood pathway. While separate regression analyses of blood lead vs soil lead and dust lead, and dust lead versus soil lead can be carried out, combining these equations without consideration of the simultaneous estimation of the equations using the same data involves a potential "simultaneous equation" bias. Therefore, simultaneous modeling of blood lead and dust lead was carried out in the Cincinnati report for each round. Some cross-sectional structural equation model analyses were performed in this report using Round 1 or preabatement data to assesss the extent to which soil might have been a significant source of dust lead and blood lead before abatement. Structural equation models using differences of blood lead and environmental lead allow

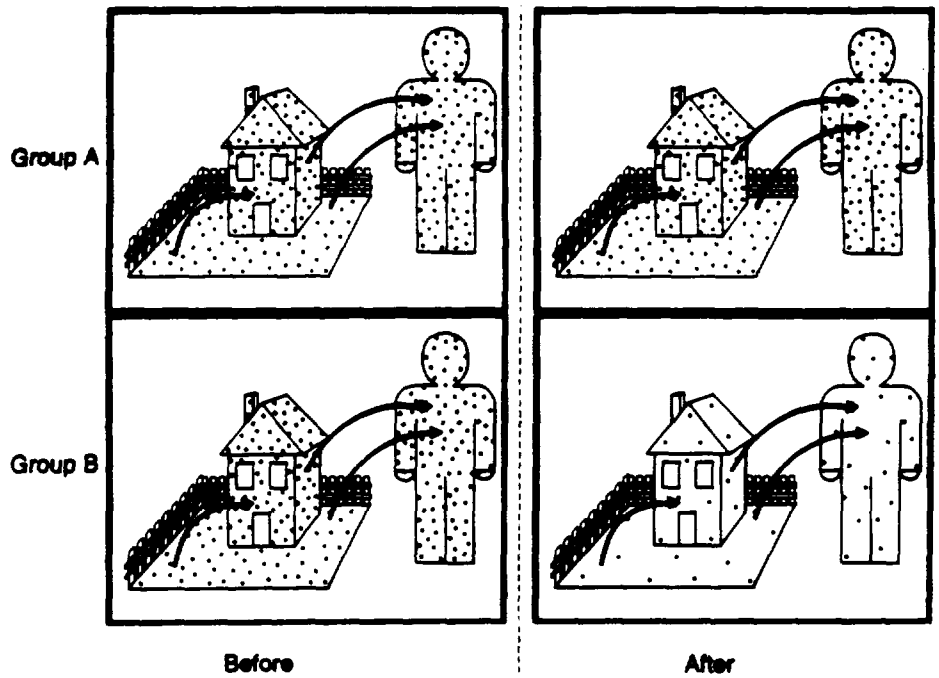

Figure 5-3. Cross-sectional structural equations model. Arrows show that the individual child blood lead concentrations are adjusted for soil lead and dust lead concentrations or loadings in the child's residence, and may also be adjusted for other household-specific or child-specific covariates such as age or gender. There are also arrows showing that house dust lead is related to soil lead, so that the total soil effect consists of a direct exposure pathway and an indirect soil-to-dust-to-blood pathway. However, blood lead is compared across treatment groups at each round with no analyses of effects between rounds.

some longitudinal structure, but these analyses typically do not include assessment of changes in blood lead and its environmental covariates.

With cross-sectional structural equation modeling, the response variable Y_{ir} depends on covariates $\mathbf{Z}_{ir}$ and X_{ir}, expanding Equation 5-5 to the form

$$Y_{ir} = G_{gr} + X_{ir}B_{gr} + Z_{ir}F_{gr} + e_{ir} \qquad (5\text{-}7)$$

where X_{ir} is related to Z_{ir}, or other covariates represented as W_{ir}, in the following manner:

$$X_{ir} = C_{gr} + Z_{ir}D_{gr} + W_{ir}L_{gr} + d_{ir} \qquad (5\text{-}8)$$

C_{gr} is the covariate group effect for group g and round r

Z_{ir} is the second covariate, interacting with X_{ir} and also directly affecting Y_{ir}

W_{ir} is the third covariate interacting with X_{ir} but not directly affecting Y_{ir}

D_{gr} is the regression coefficient for Z_{ir}, and

d_{ir} is the measurement error term for covariate X_i at round r

Note that Z_{ir} has both a direct and indirect effect on Y_{ir}. The direct effect is as a component of the expression $Z_{ir}F_{gj}$ in Equation 5-7, and the indirect effect is as a component of X_{ir} in Equation 5-7, as shown in Equation 5-8. With this method, Equations 5-7 and 5-8 would be solved simultaneously in an iterative manner to estimate the value of the regression coefficient that provides the best fit for the combined system of equations. In performing these calculations, we used several structural equation model calculation algorithms to obtain the best fit. These algorithms are discussed in Section 5.6.

The output of the cross-sectional structural equation model provides several types of treatment group response sizes:

Response to treatment group (base)	$G_{1r} - G_{2r}$	(5-9)
Difference between covariate intercepts	$C_{1r} - C_{2r}$	(5-10)
Difference in response to covariate X	$B_{1r} - B_{2r}$	(5-11)
Difference in direct response to covariate Z	$F_{1r} - F_{2r}$	(5-12)

There is also a measure of the total (direct and indirect) relationship of Z to Y, expressed as

$$F_{gr} + B_{gr}D_{gr} \qquad (5\text{-}13)$$

and a difference in total relationship of Z to Y, expressed as

$$(F_{ir} + B_{ir}D_{ir}) - (F_{2r} + B_{2r}D_{2r}) \qquad (5\text{-}14)$$

The output from this model is an estimate of the transfer from one compartment to another (e.g., for soil to dust, 0.2 μg Pb in dust per μg Pb in soil). Structural equation models are normally visualized as boxes and arrows, with the boxes representing environmental components, such as house dust, and the arrows representing transfer between

boxes. This method establishes that specific pathways exist in the child's exposure environment.

Cross-sectional structural equation models were used in the Cincinnati report and in this assessment to examine the pre-abatement environmental lead exposure pathways. While the population samples of the three studies were chosen for use in a longitudinal intervention study, and not for the purpose of assessing baseline exposure in the communities, the baseline data nonetheless represent a useful snapshot of children in certain neighborhoods in Baltimore, Boston, and Cincinnati. The limitations of this approach are that the study populations may not have been a fully representative sample of the true neighborhood populations, especially in Boston and Baltimore. The impact of these limitations is discussed in greater detail in Section 5.4.

5.1.2.4 Longitudinal Analysis of Covariance

The next four methods are longitudinal analyses and, as such, are more appropriate for these studies. These analyses evaluate changes in childhood blood lead and in environmental lead exposure pathways subsequent to (and by inference, as a result of) soil lead or dust lead interventions. Among the possibilities considered are: (1) blood lead decreases as a result of a direct change in environmental exposure to soil; (2) blood lead decreases as a result of both a decrease in soil lead and a decrease in dust lead, where the decrease in dust lead load may be by dust intervention or by the impact of soil intervention on dust lead load; (3) blood lead decreases as a result of changes in dust ingestion as a result of other interventions, such as a decrease in dust loading (cleaner house means less dust ingested) or changes in the child's behavior (parental education) that decrease dust ingestion; and (4) blood lead changes as a result of factors unrelated to intervention, including growth and normal changes in child behavior.

Longitudinal analysis of covariance (Figure 5-4) is similar to cross-sectional analysis of covariance in that it has fixed treatment groups with continuous or categorical covariates, but the response variable is also adjusted for the blood lead concentration from the previous round (Y_{ir-1}). Figure 5-4 is similar to Figure 5-2, but with an additional arrow to show that blood lead is now controlled or adjusted for the preabatement blood lead concentration in the

difference between treatment groups in the response variable, but explicitly includes pretreatment levels of response. Similar to cross-sectional analysis of covariance, however, this method also assumes the pretreatment level of response is perfectly known and that the covariates are known without measurement error (see Section 5.1).

The units of the response term are expressed as a ratio of the response to the covariate. For example, the Boston group used this method to estimate a range of response to intervention as being a decrease of 0.7 to 1.2 μg/dL for each incremental decrease of 1000 μg/g Pb in soil during the first phase of their study, and a somewhat larger decrease during the second phase.

5.1.2.5 Repeated Measures Analysis of Variance

Two of the remaining methods are repeated measures analyses, and one is a structural equation modeling method. Repeated measures analyses evaluate whether intervention affected the child's blood lead, whereas structural equation models assess whether this change can be attributed to the soil-dust-blood pathway. The two approaches are complementary, especially for a longitudinal intervention study.

Repeated measures analysis of variance contains a group effect term (G_{ir}), a household effect term ($H_{h(g)}$), and an individual child effect term ($I_{i(gh)}$), in addition to the error term.

$$Y_{ir} = G_{gr} + H_{h(g)} + I_{i(gh)} + e_{ir} \qquad (5\text{-}16)$$

5.1.2.6 Repeated Measures Analysis of Covariance

Similarly, repeated measures analysis of covariance adds the covariate term $X_{ij}\ B_{gi}$ that first appeared in Equation 5-5.

$$Y_{ir} = G_{gr} + X_{ir}B_{gr} + H_{h(g)} + I_{i(gh)} + e_{ir} \qquad (5\text{-}17)$$

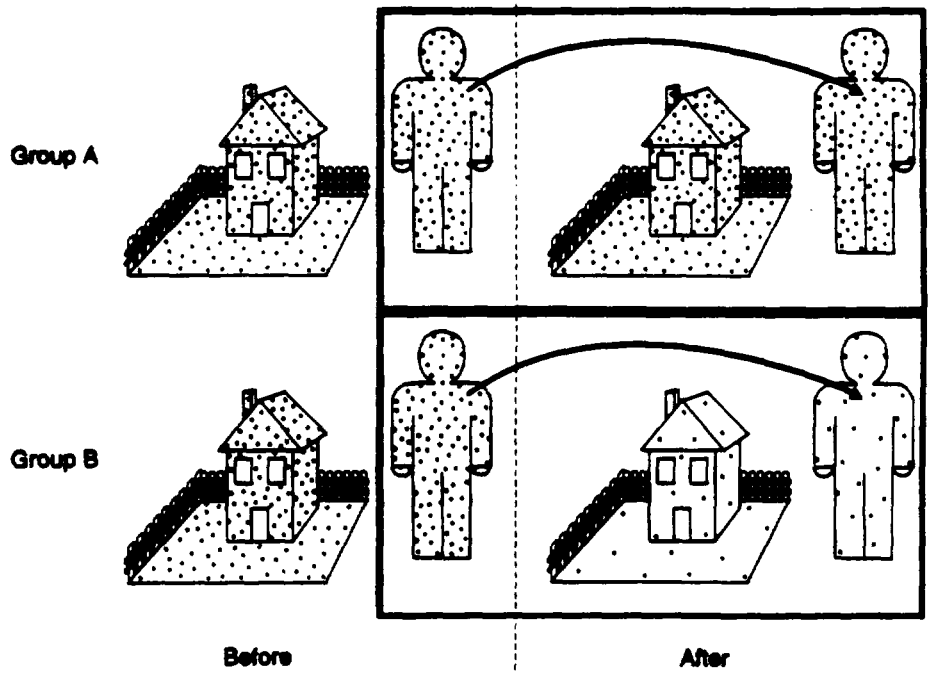

Figure 5-5. Repeated measures analysis of variance. The arrow from one child figure to the next shows that blood lead at the postabatement round is adjusted for preabatement blood lead for each individual child. Blood lead is compared across treatment groups across different rounds for children in the study at each round.

transport of lead from soil. The LSEM methods in this report are used in order to evaluate some of the possible mechanisms by which soil abatement may operate, which was not required in the individual study reports. The LSEM analyses carried out here also allowed evaluation of the sensitivity of the repeated measures ANOVA and ANCOVA analyses to an alternative modelling approach.

5.1.2.7 Longitudinal Structural Equation Models

Longitudinal structural equation model equations (Figure 5-7) are similar to the cross-sectional form for structural equation models, with the addition of terms for the influence of previous rounds, expressed with subscript r-1:

$$Y_{ir} = G_{gr} + X_{ir}B_{gr} + Z_{ir}F_{gr} + Y_{ir-1}A_r + e_{ir} \qquad (5\text{-}18)$$

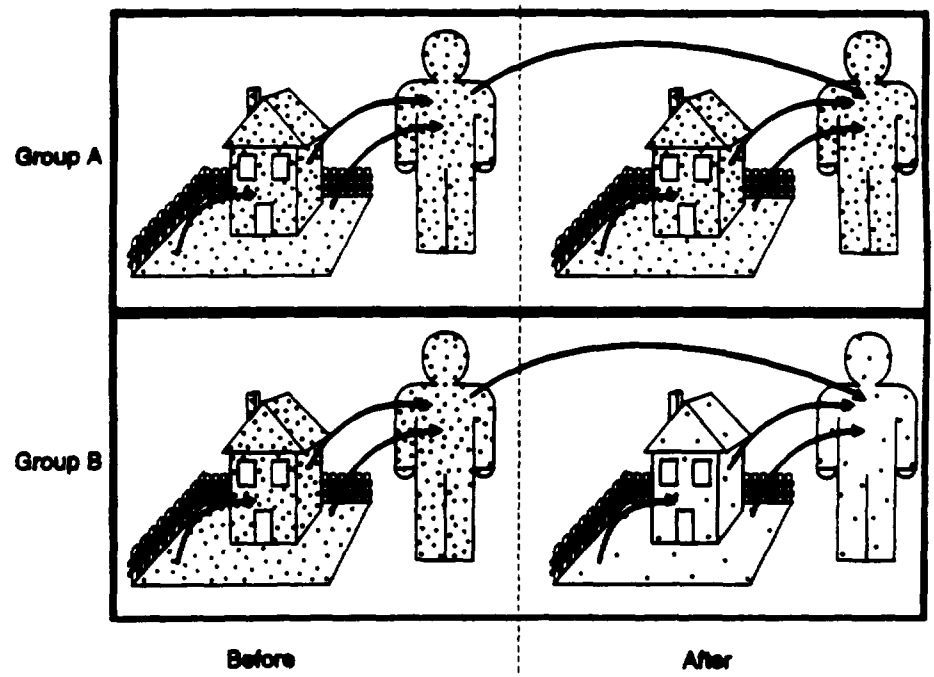

Figure 5-7. Longitudinal structural equation model. The arrow from one child figure to the next shows that blood lead at the postabatement round is adjusted for preabatement blood lead for each individual child. Blood lead and dust lead are adjusted for covariates simultaneously at each round to eliminate simultaneous equation bias. Postabatement blood lead is adjusted for preabatement blood lead.

5.1.3 Specific Problems with Statistical Methods

A potential problem arises in simple comparisons of group mean values during a longitudinal study when different individuals are present at different phases of the study. For example, some individuals in the preabatement phase of the study may have dropped out by the time of the postabatement phase, whereas other individuals who were not in the preabatement phase may have been recruited into the postabatement phase (e.g., infant siblings who reached enrollment age status during the study). Although it would be reassuring to think that attrition and recruitment do not depend on the treatment group, and that children lost or gained during the progress of the study are no different from those enrolled throughout the study, this cannot be guaranteed. One of the simplest solutions is to limit the analyses to children who were present during all key phases of the study.

preabatement − postabatement blood lead < fraction of preabatement blood lead.

This suggests that a better index for abatement effectiveness might be a partial difference:

postabatement − (1 − fraction) preabatement blood lead > 0.

Unfortunately, the value of this fraction is not known well enough to define a priori the partial difference for use as an index of lead effectiveness, because the value of the retained fraction of lead depends on the time since abatement, the child's age, and probably other factors including the initial blood lead level.

Even though statistical models could be based on the partial differences of blood lead levels between pre- and postabatement phases, the environmental exposure variables are themselves more or less correlated with earlier measurements of the exposure variables. This violates one of the most important assumptions about linear regression models, and generally about linear models such as the analysis of variance and the analysis of covariance. That assumption is that the predictor variables or regressors are known without statistical error. Although the statistical error is usually called "measurement error" (Fuller, 1987), the errors include many other kinds of variability. In environmental epidemiology, the most common measurement errors in exposure include behavior or activity pattern variability, repeat sampling variability, sampling location variability, as well as analytical error. That is, the observed value of the predictor, such as floor dust lead loading, may not perfectly reflect the activity of the child and the child's actual exposure to dust lead over time.

One way to deal with this is to predict the precursor exposure variables in an environmental model. For example, suppose that blood lead is predicted by hand lead, soil lead, dust lead, and by a preceding value of the blood lead. Hand lead may then be predicted by current dust and soil lead levels, and dust lead by current soil lead, so that in addition to the direct effect of soil lead on blood lead, there are indirect effects from soil to dust to hand to blood, and from soil to hand to blood. This approach allows estimation of the measurement error variance in the precursor lead exposure variables in terms of residual deviations between the observed exposure variable and its best estimate from its own precursors. If the model is correct, this approach will essentially eliminate the bias

and blood lead independently. In the second form, blood lead data alone are plotted by round and age for each individual child. This method gives some perception of individual differences in blood lead concentrations and individual differences in response to intervention. Each of these two visual presentations has limitations that should discourage the reader from drawing conclusions about the impact of intervention. These limitations are the same whether blood leads are characterized by the group mean, geometric mean, median or other percentile values. The first is that some of the children in any treatment group are probably not exactly the same children at one phase of the study as at a subsequent phase. Some children will almost certainly be lost to follow-up by moving or by refusal to participate (normal processes of attrition in longitudinal studies), whereas other children may be added by recruitment (such as at Round 3 in the Baltimore study) or as additional members of households where other children are already enrolled in the study. Since children who are lost to follow-up or who are added to the study may differ in some systematic ways from children who were retained throughout the study, it may be prudent to analyze data from these children who were not present separately from those who were present at all relevant phases. On the other hand, if study results are restricted only to children who were present at certain specific pre- or postabatement phases of the study, then repeated measurements on the same child at different phases of the study are not statistically independent of each other. Although data from one treatment group at a given phase are independent of data from a different group, data on the same group at a different phase are not independent of data from an earlier phase.

In the Boston study analyses, the same subset of children was used here as in the Boston report, excluding the same two children who had become lead-poisoned. For the Baltimore data, the small group of participants from the treatment group whose properties were not abated were assigned to a separate control group, rather than merging them with the main control group. The Cincinnati neighborhoods are treated here as individual study groups and include all children recruited (both rehab and nonrehab), except for the four children who were undergoing treatment for lead poisoning.

The presentation of these group mean data, illustrated in Figure 5-9, uses a similar format for all of the figures in this series. Each treatment group is represented in each round by a box and whisker plot. Each box has a mark approximately midway that shows the

and 5-12. These data represent the percentile calculated from the individual parcel means of several individual soil samples. They show, for all three studies, a substantial reduction in the amount of lead in abated soil areas. In Boston and Cincinnati, where follow-up soil measurements were taken, this reduction persisted for the duration of the study. In Baltimore, the postabatement measurements were made only in the locations where soil had been excavated and removed.

Each study was able to achieve the targeted concentration for abated soil. The median soil concentrations following abatement are not substantially higher than the specifications for clean soil. The amount of soil lead reduction actually achieved directly influences the expected changes in dust lead and blood lead. Soil lead concentrations vary widely over relatively small distances. Because it was not feasible to return to the exact spot each time for sequential soil samples, two sequential samples from the same plot may vary widely.

The median of the soil parcel means for the Boston and Cincinnati studies show that abated soil concentrations [BOS SPI, CIN SEI(P), CIN I-SE(D), and CIN I-SE(F)] dropped substantially after abatement (Figures 5-10 and 5-11) whereas unabated soil (BOS PI, BOS P, and CIN NT) showed virtually no change.

5.2.2 Changes in Exterior Dust Concentrations and Loadings

In Cincinnati, exterior street and sidewalk dust concentrations remained relatively constant throughout the study (Figures 5-13 and 5-14) and are much higher than the soil concentrations, suggesting a source or sources with higher lead concentrations than soil that mix with leaded dust from soil to form exterior dust. A possible conclusion is that sources of lead in exterior dust, other than soil, impacted each neighborhood or groups of neighborhoods, differently. This is reasonable because the neighborhoods are geographically separated. Five of the neighborhoods (Back, Dandridge, Findlay, Mohawk, and Pendleton) are nearly contiguous and lie in a larger neighborhood known locally as "Over-the-Rhine". The sixth neighborhood, Glencoe, lies approximately ½ mile away. Interpretation of the spatial distribution of the Cincinnati data within each neighborhood is not possible without more information on the location of the dust samples.

For Boston and Baltimore, the question arises that there may also be external sources of lead other than soil that contribute to household dust and to the exposure of children during

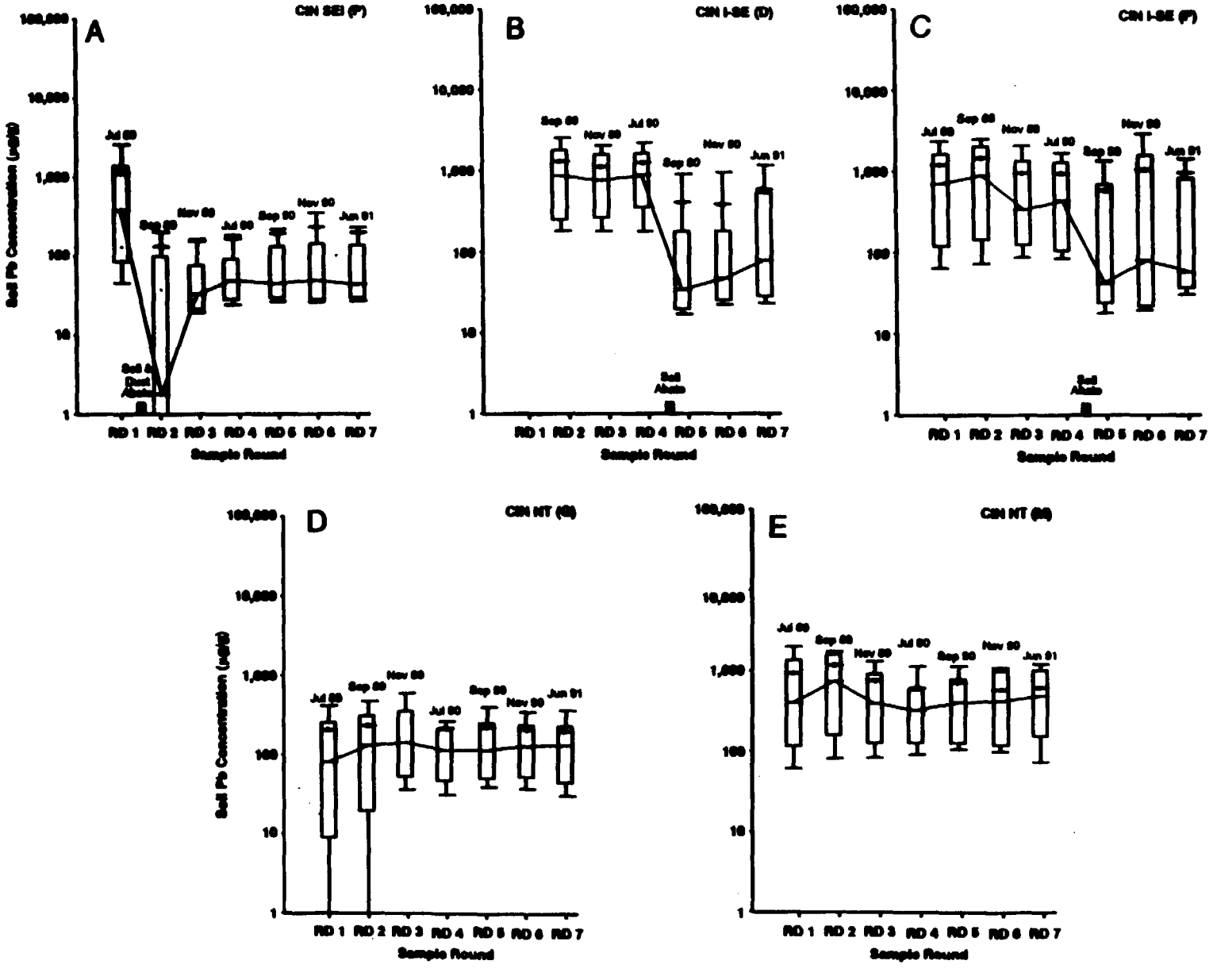

Figure 5-11. Cincinnati soil lead concentrations (l scale). Data are shown by neighborhood and reflect abatement in the first or second year of the study. Th vere no soil samples taken in the ndridge neighborhood (Panel B)

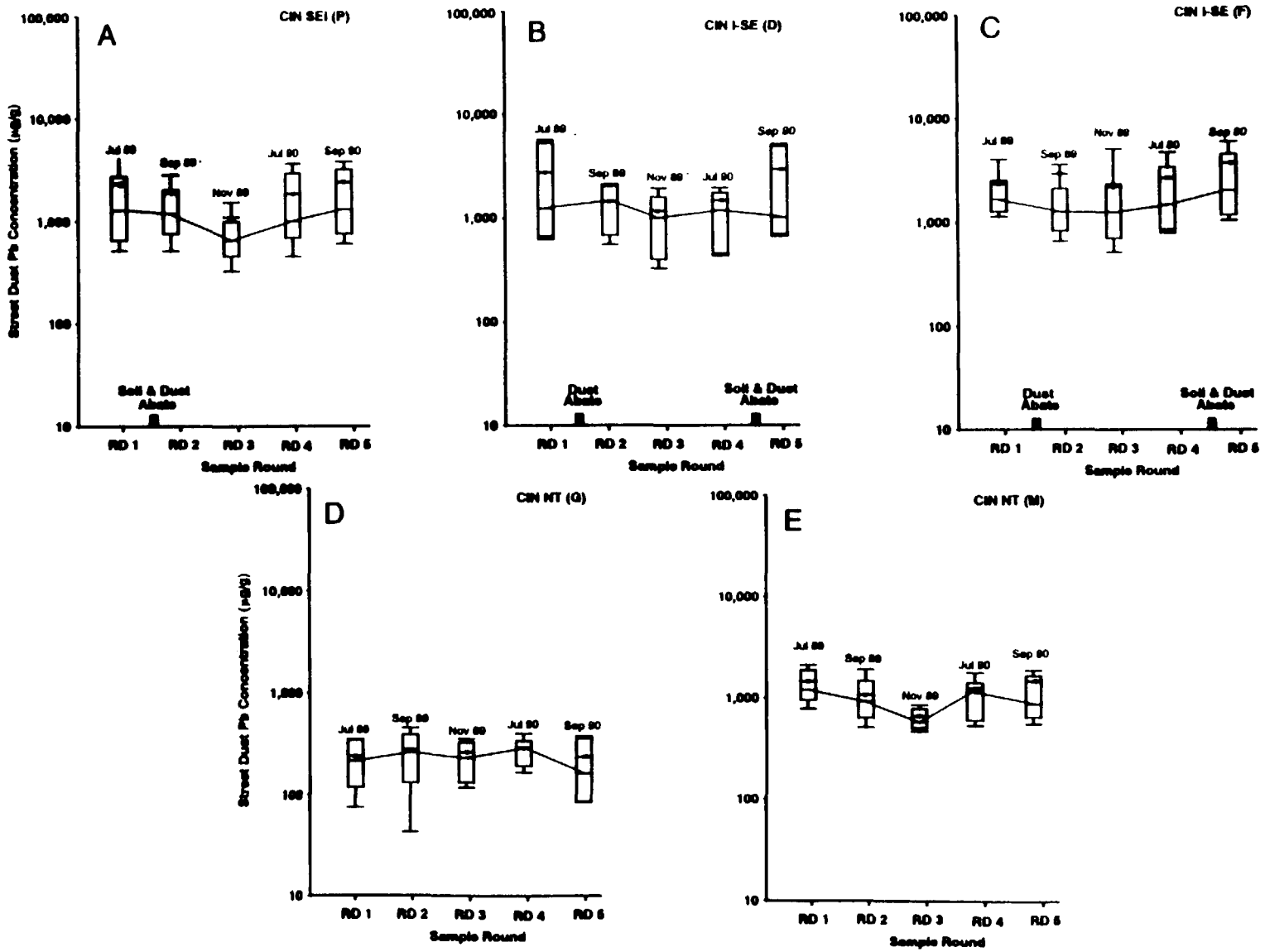

Figure 5-13. Exterior dust lead concentrations (log scale) from the street samples in the Cincinnati study. Data are by neighborhood. Exterior dust samples were not reported for rounds 6 and 7.

5.2.3 Changes in Interior Dust Concentrations and Loadings

Interior dust is measured in both concentration and surface loading. Concentration is measured in micrograms of lead per gram of dust, whereas loading is measured in milligrams of lead per square meter. When dust abatement is performed, the amount of dust changes, but the concentration of lead in the dust does not. Therefore, there should be no change in dust lead concentration unless the source of the dust changes. Where soil abatement has been performed in connection with dust abatement, the dust lead concentration should also decrease abruptly if the soil is the major component of the dust. If there is a mixture of dust sources and only one has been abated, the lead concentration would change less abruptly, according to the contribution from each source.

The data for the Boston study interior dust measurements are shown in Figures 5-15 through 5-20. The high concentrations of lead in individual measurements of window well dust (5,000 to 22,000 μg/g) indicate the possible presence of lead-based paint (Figure 5-15).

The Cincinnati study (Figures 5-21 through 5-23) found relatively constant dust lead concentrations during the first year (Rounds 1-4). Data are not available for Rounds 5-7. Data for window wells are shown in Figures 5-24 through 5-26 and entry ways, Figures 5-27 through 5-29. The window well concentrations were lower in Cincinnati (1,000 to 2,300 μg/g) than in Boston, suggesting a minimum influence of lead-based paint.

5.2.4 Changes in Hand Dust Lead Loadings

Because hand-to-mouth activity is one route by which lead may be ingested, the amount of lead on the child's hand is an indicator of exposure. The hand wipe data for Boston are in Figure 5-30, for Baltimore, in Figure 5-31, and for Cincinnati, in Figure 5-32. Only lead loading information is available because there is no measure of the amount of dust removed. The units of measurement are micrograms per pair of hands rather than micrograms per square meter. This is an important link in the exposure pathway that measures actual contact with the child's dust environment. Hand lead loadings were expected to respond more quickly to environmental changes than blood lead concentrations.

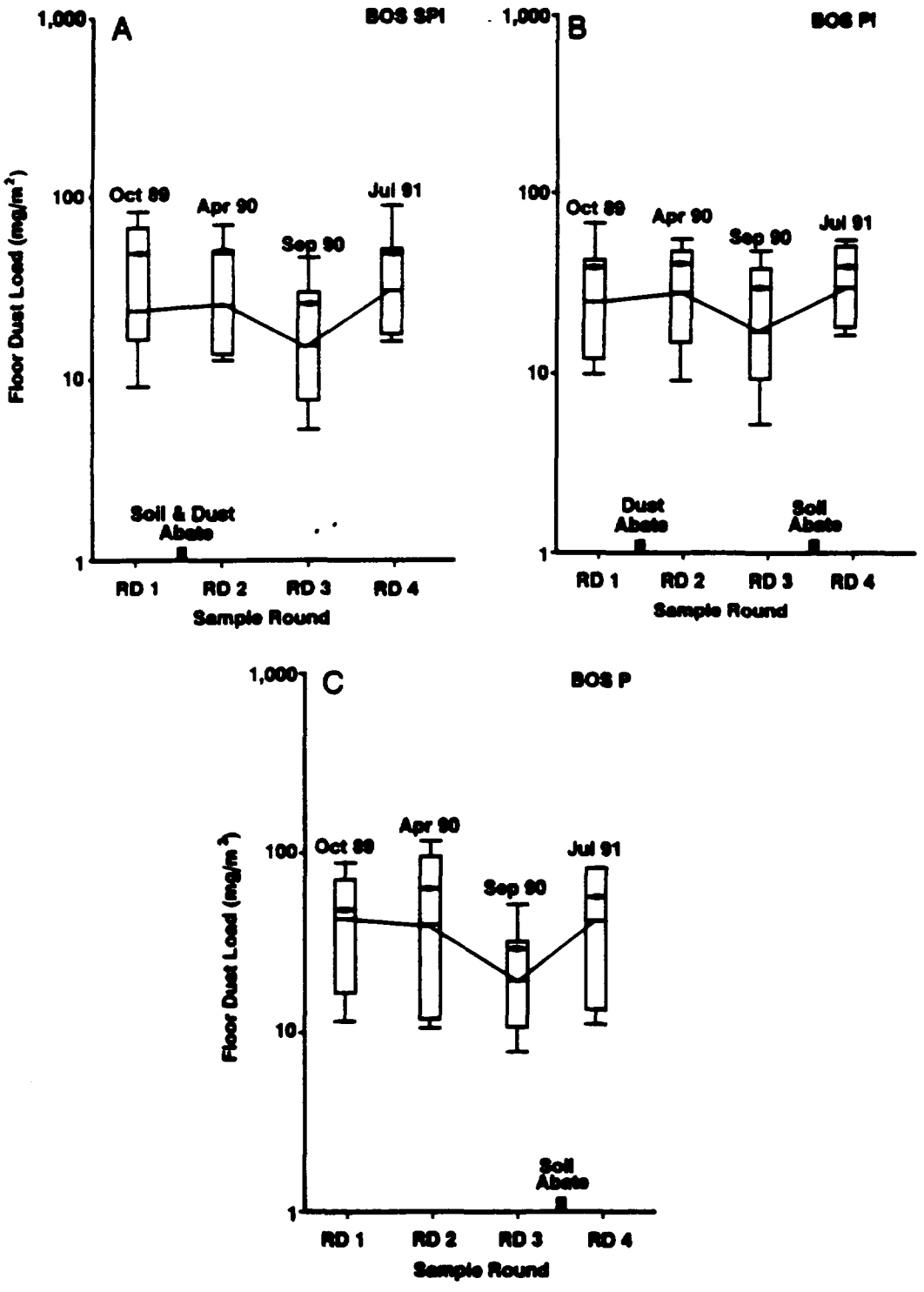

Figure 5-16. Boston floor dust load (log scale). The absence of a decrease following interior dust abatement in the BOS SPI and BOS PI-S groups suggest that house dust loadings may be replenished back to preabatement levels in a time period shorter than the interval between Round 1 and Round 2.

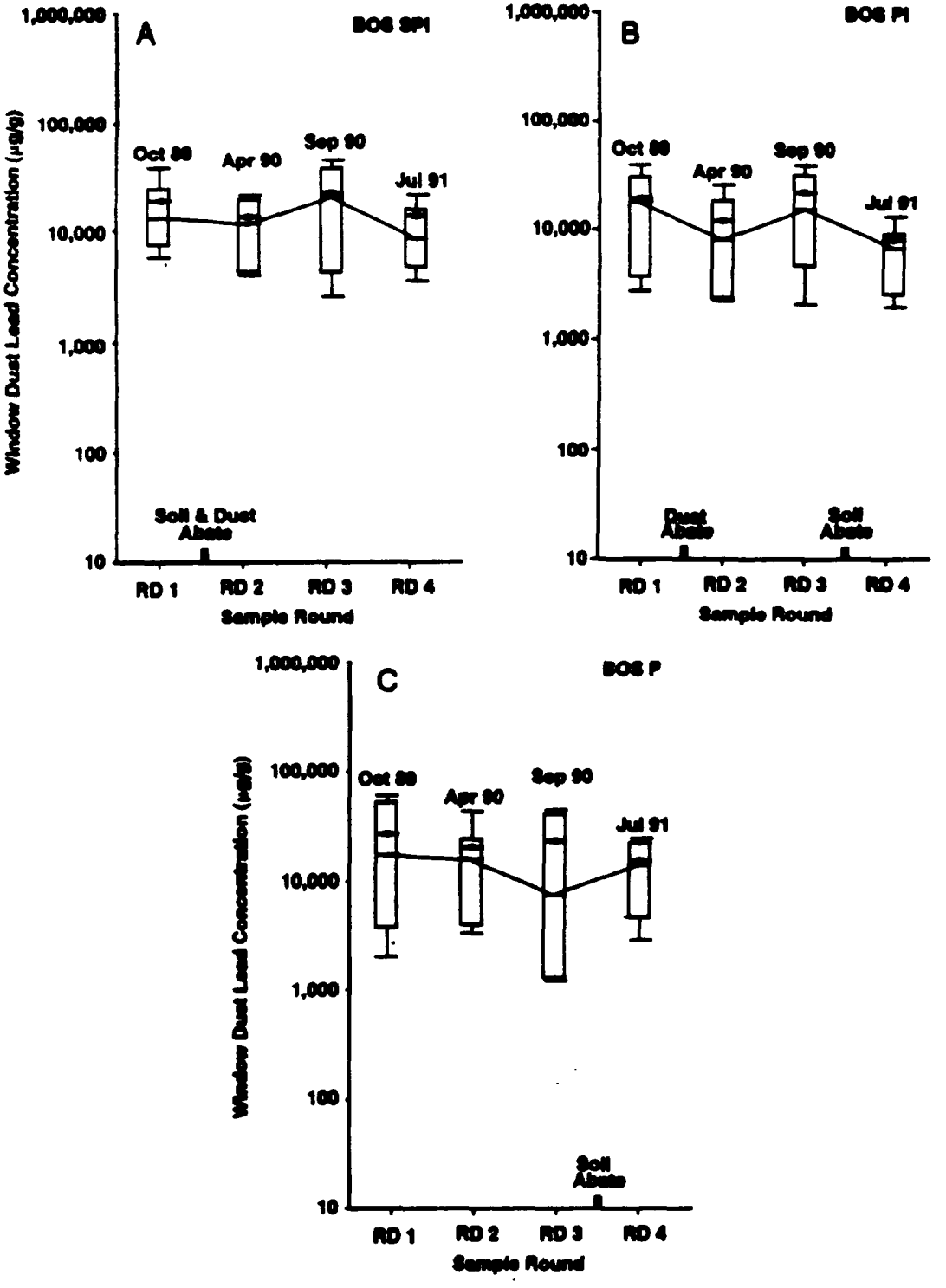

Figure 5-18. Boston window dust lead concentrations (log scale). Paint stabilization and soil abatement appear to have been effective and persistent for several hundred days, similar to floor dust. The recovery observed between April and July 1990 was not observed for the floor dust load data.

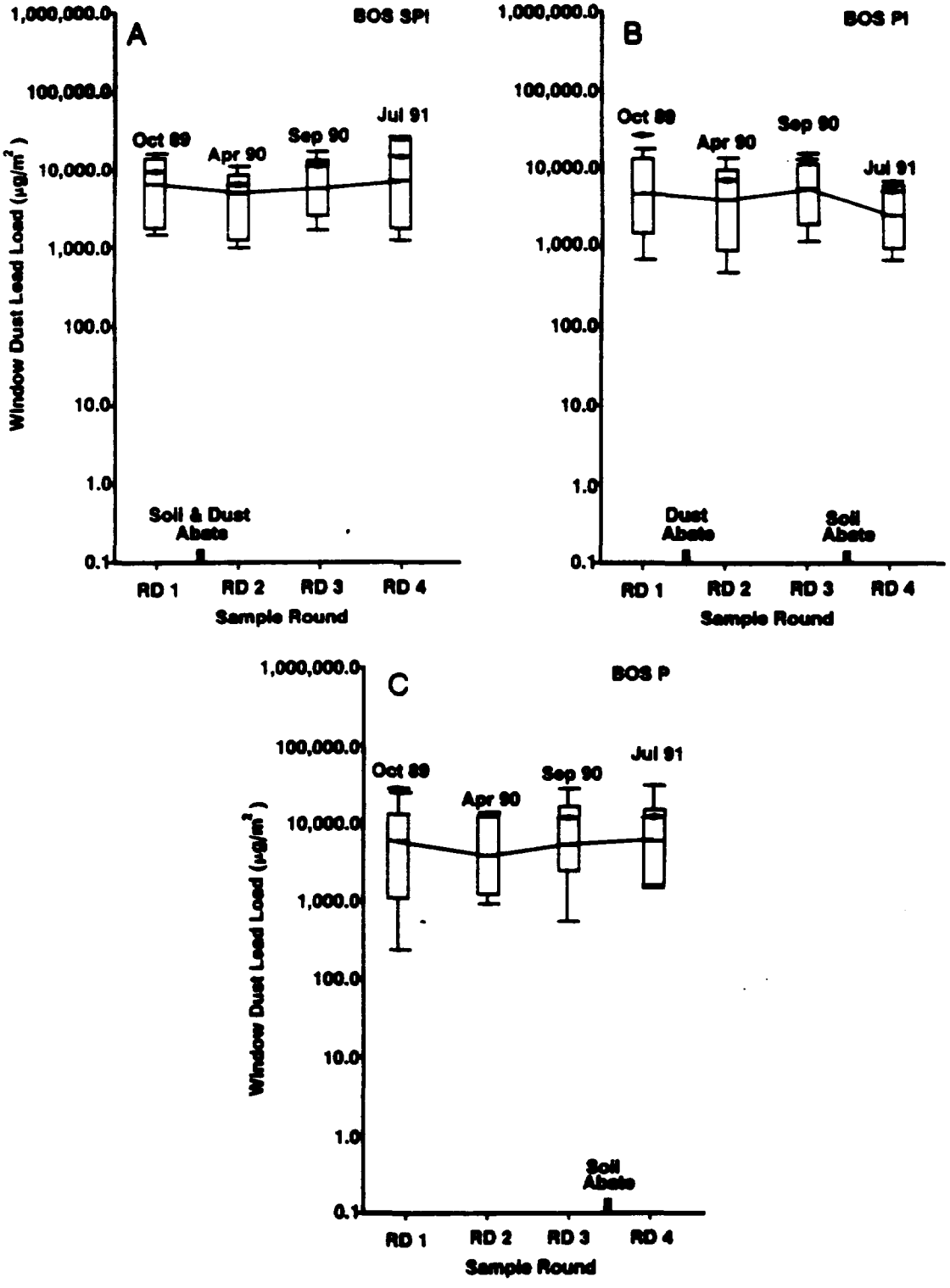

Figure 5-20. Boston window dust lead load (log scale). As with floor dust lead loads, the window data indicate that both paint and soil sources of lead were interrupted, at least temporarily. The data appear to be consistent with Figure 5-17.

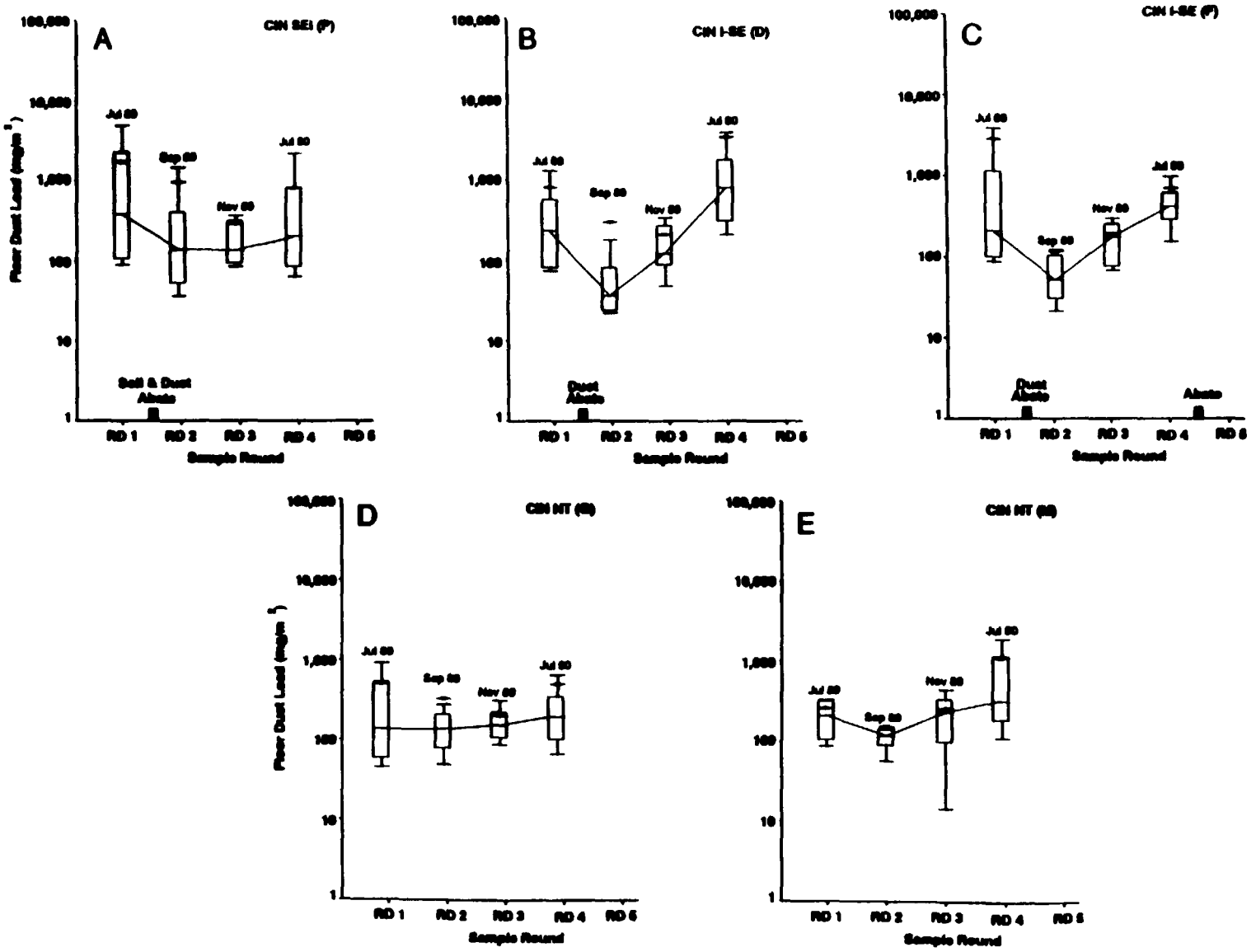

Figure 5-22. Cincinnati floor dust load (log scale). These data confirm the effectiveness of the household dust abatement and show that this reduction was persistent for as much as 60 days.

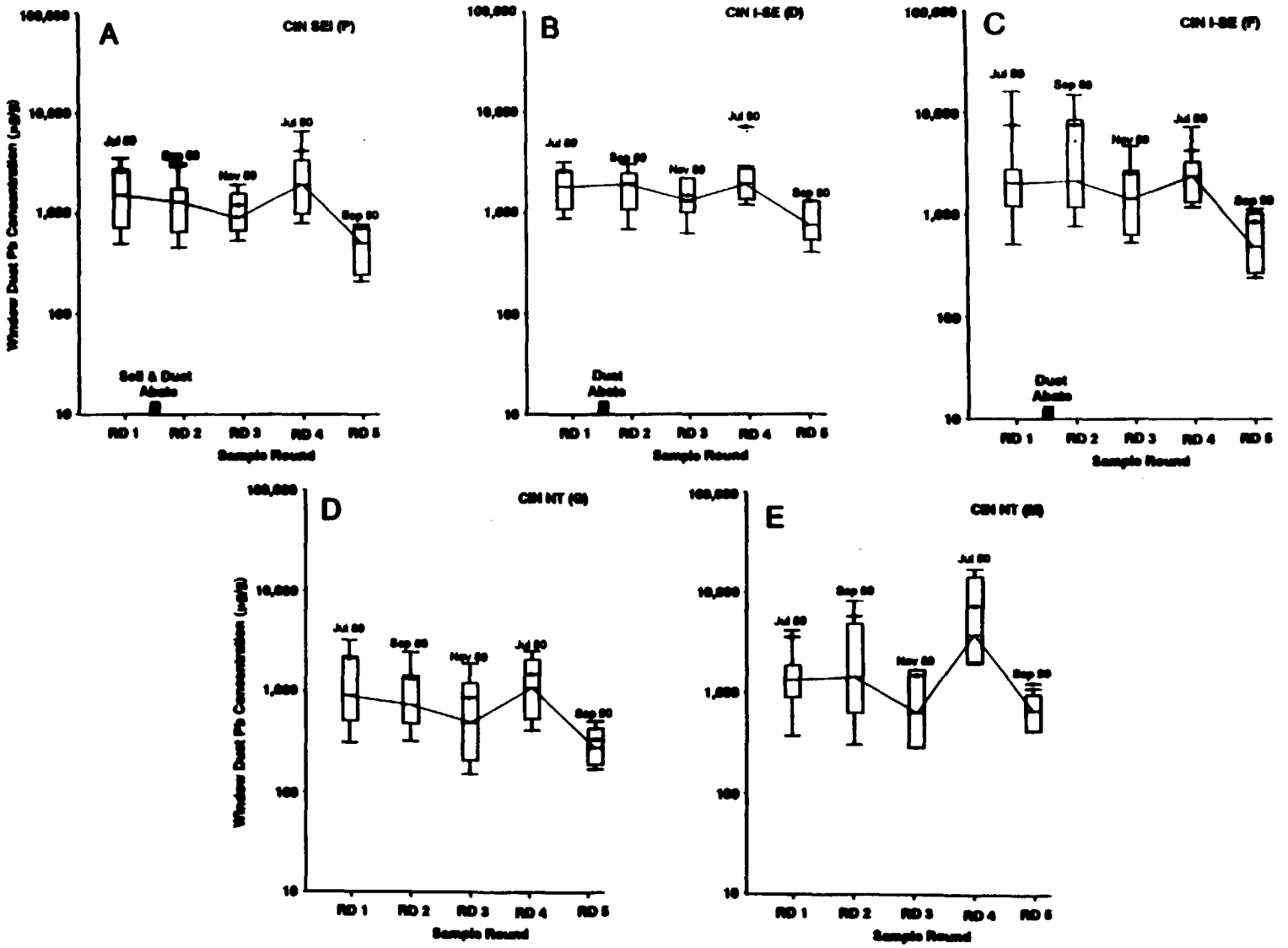

Figure 5-24. Cincinnati window dust lead concentration (log scale). The small response in lead concentration to soil and/or dust abatement appears to be consistent with the observations of the floor dust in Figure 5-21.

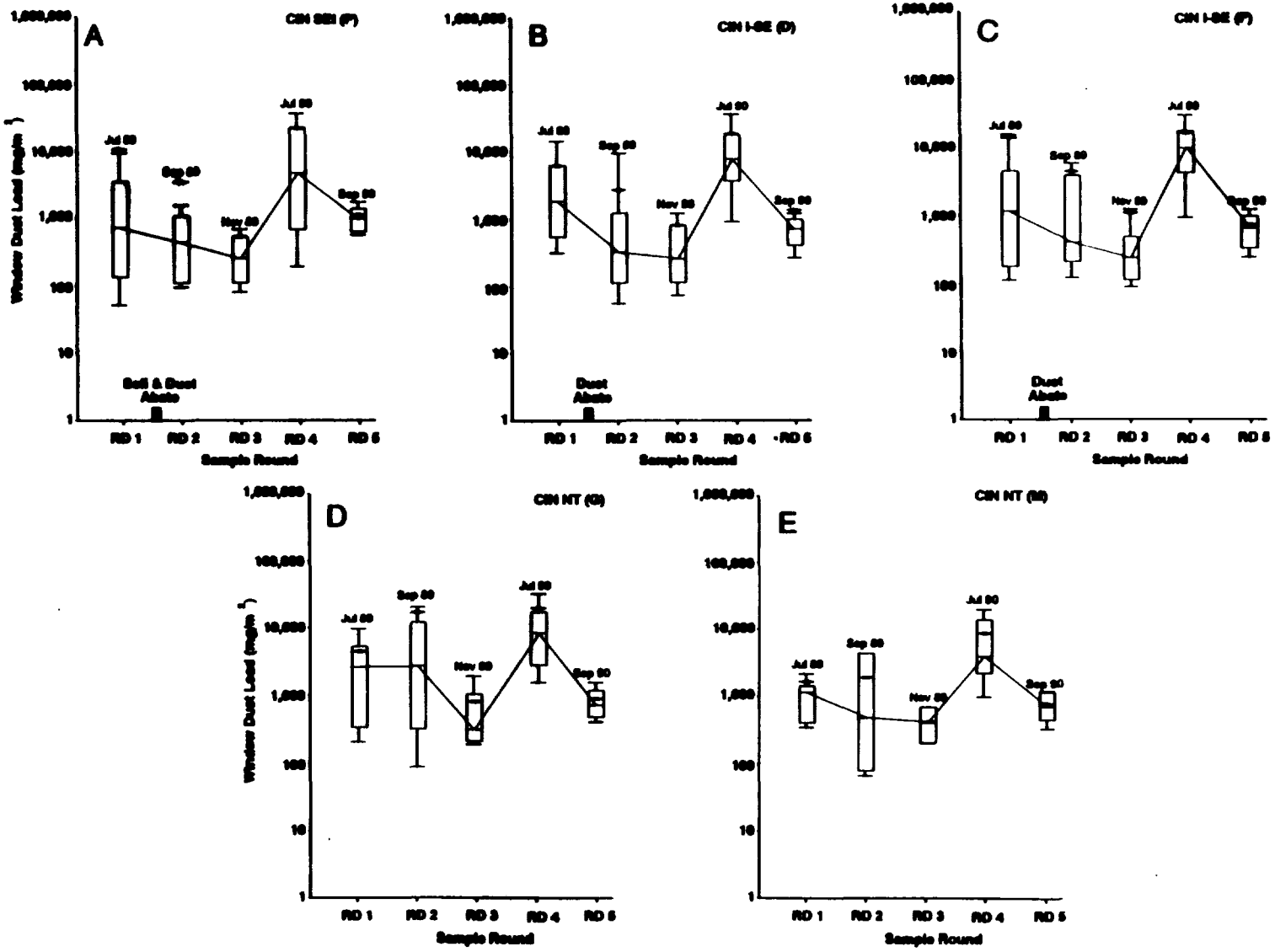

Figure 5-25. Cincinnati window dust load (log scale). The impact of abatement and the changes in the CIN NT groups are consistent between floor dust load (Figure 5-22) and window dust load as shown here.

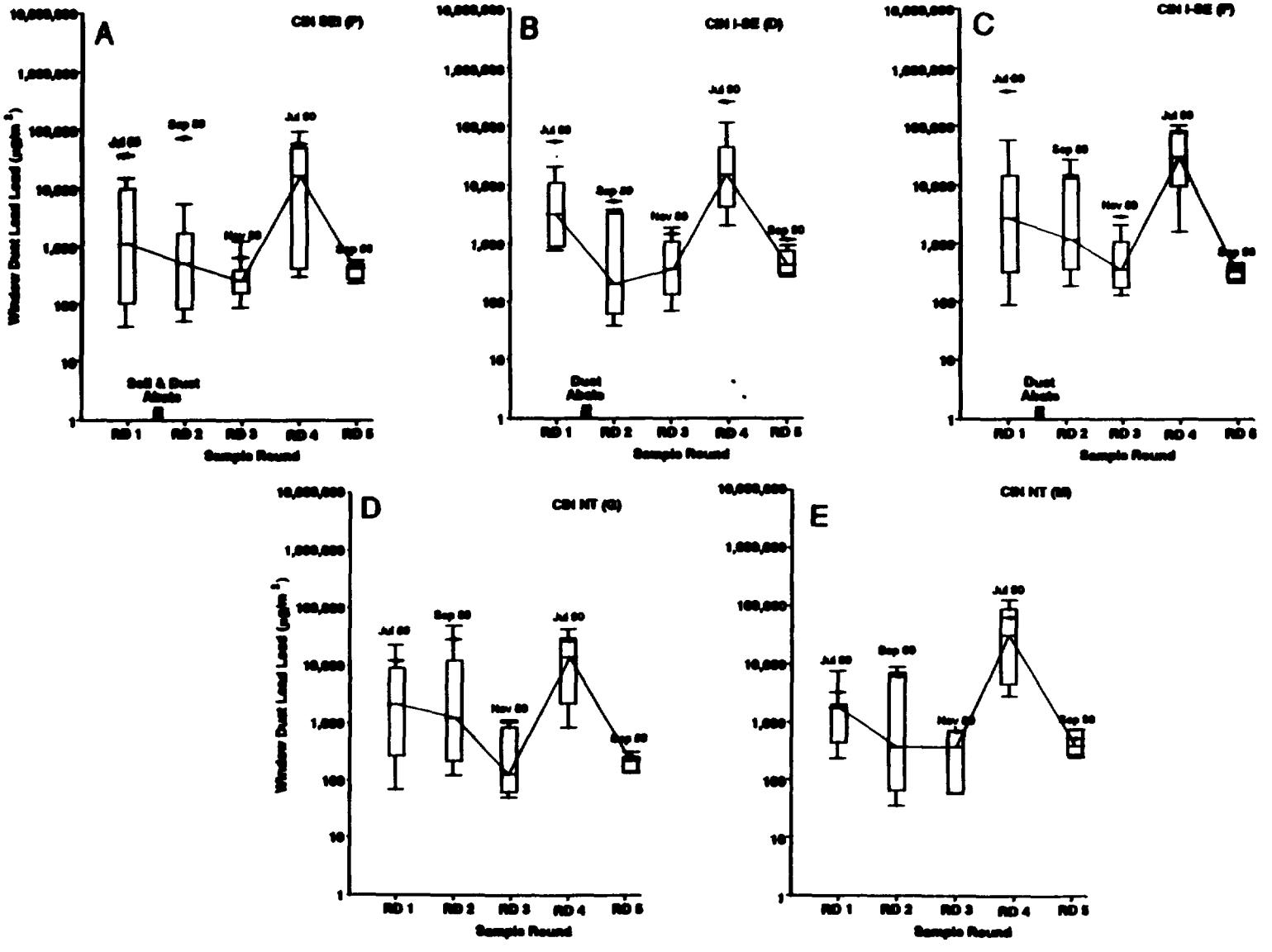

Figure 5-26. Cincinnati window dust lead load (log scale). The sharp increase between RD 3 and RD 4 may be due more to an increase in overall dust load (Figure 5-25) than in dust lead concentration (Figure 5-24).

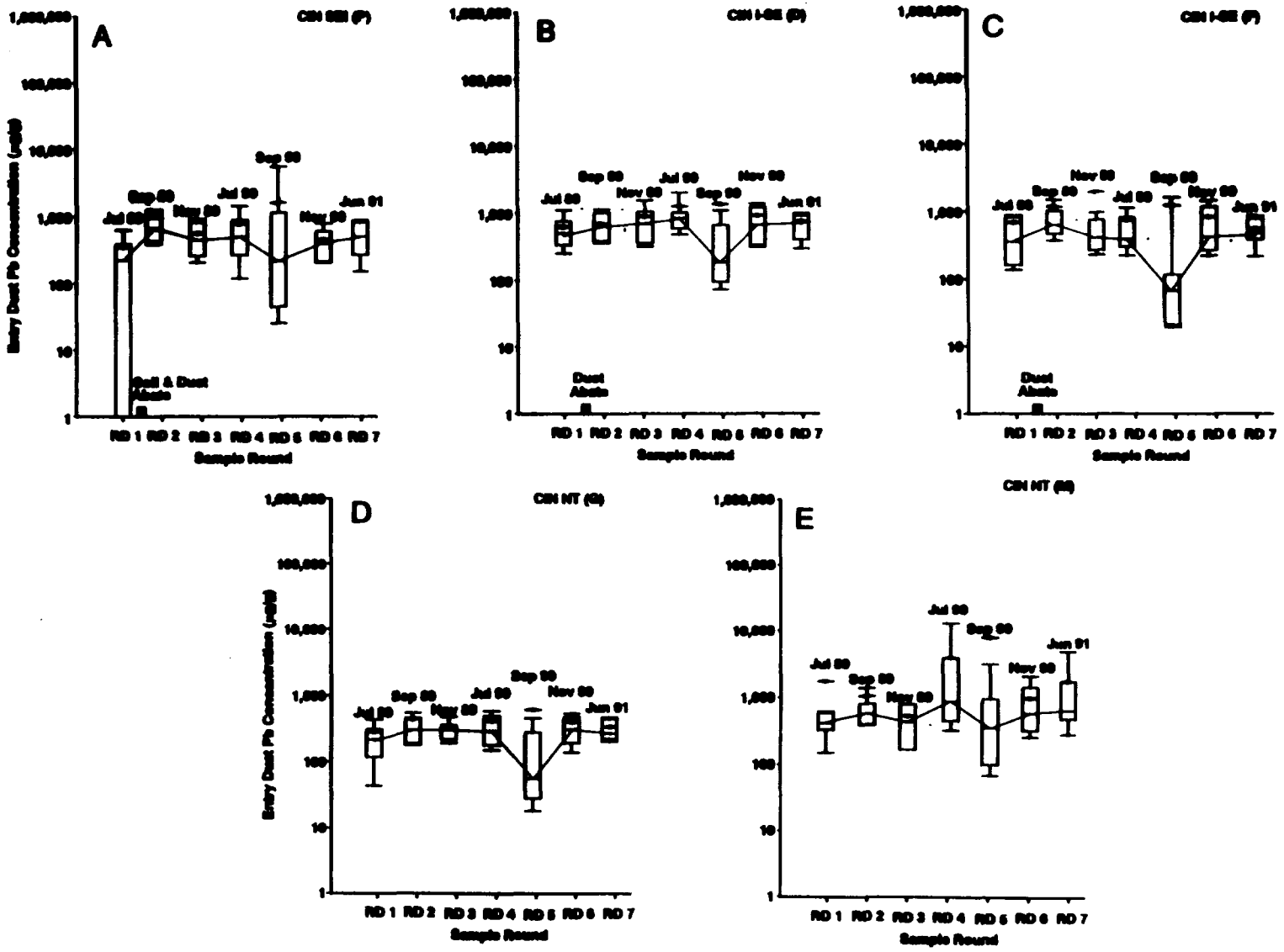

Figure 5-27. Cincinnati entry dust lead concentration (log scale). The entry way subset of the floor dust shows a pattern different from the complete floor dust data of Figure 5-21. Note the three additional rounds, September 1990, November 1990, and June 1991.

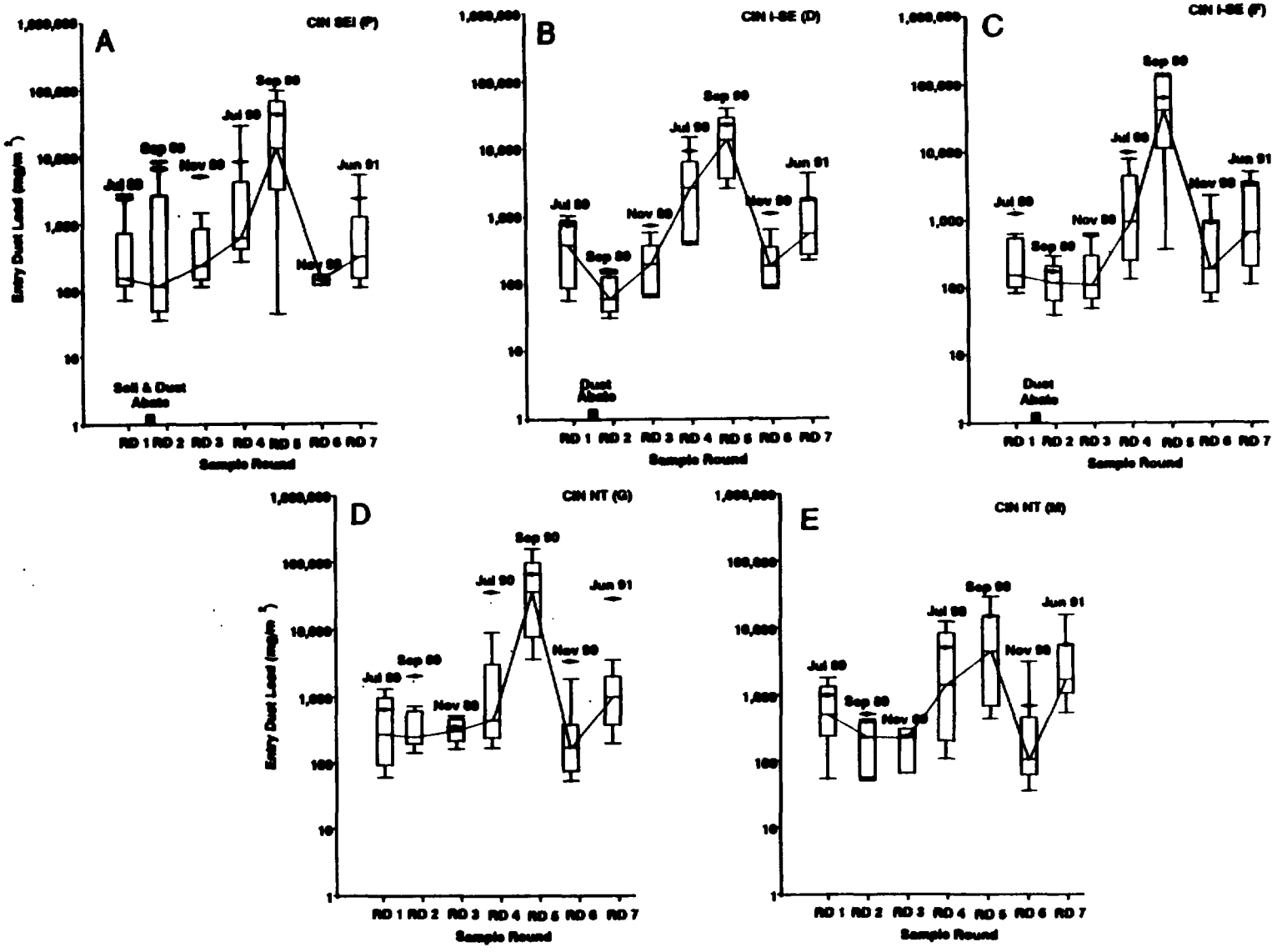

Figure 5-28. Cincinnati entry dust load (log scale). Similar to Figure 5-22, dust abatement at the entry appears to have been effective and persistent through November 1989.

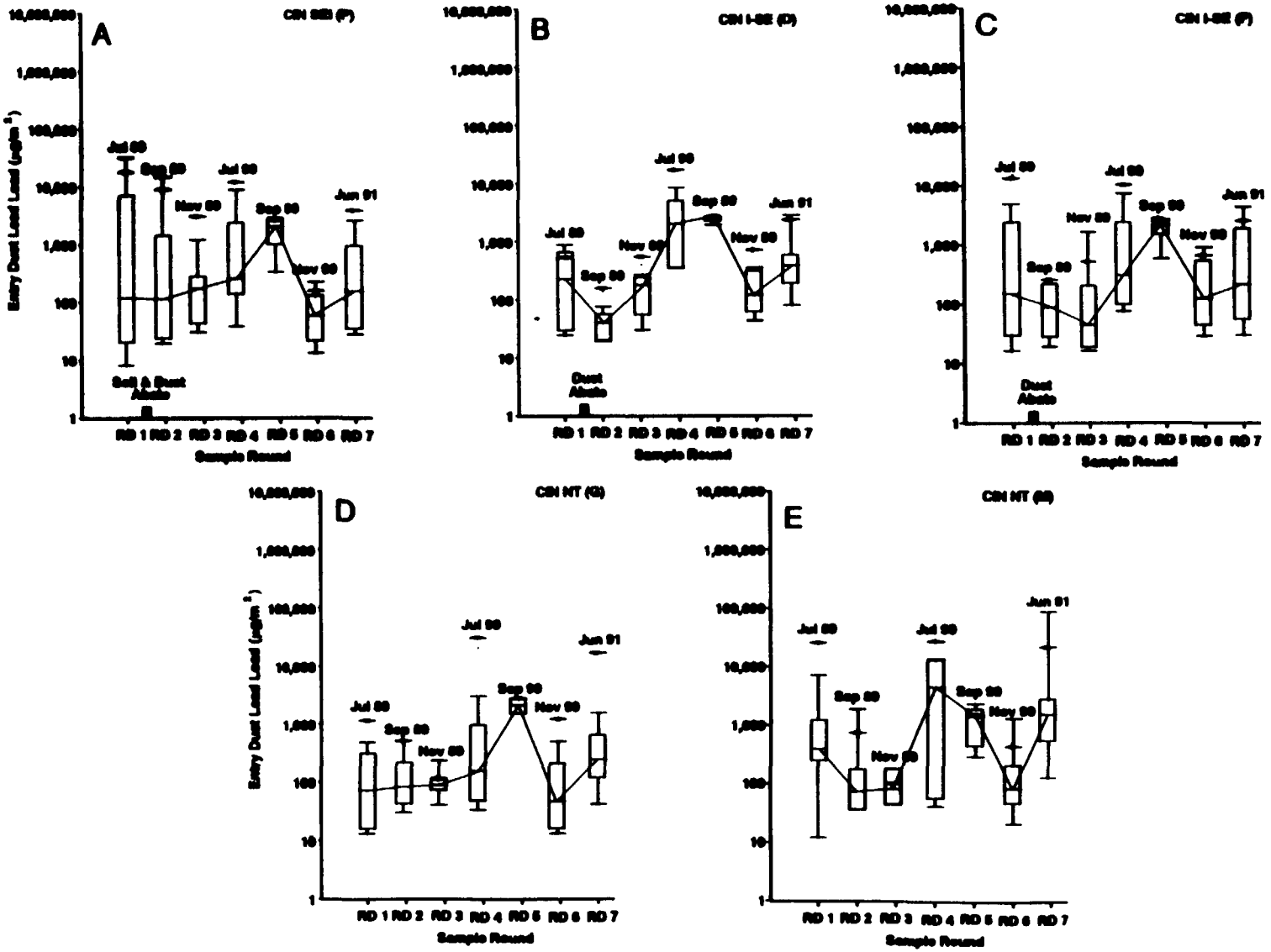

Figure 5-29. Cincinnati entry dust lead load (log scale).

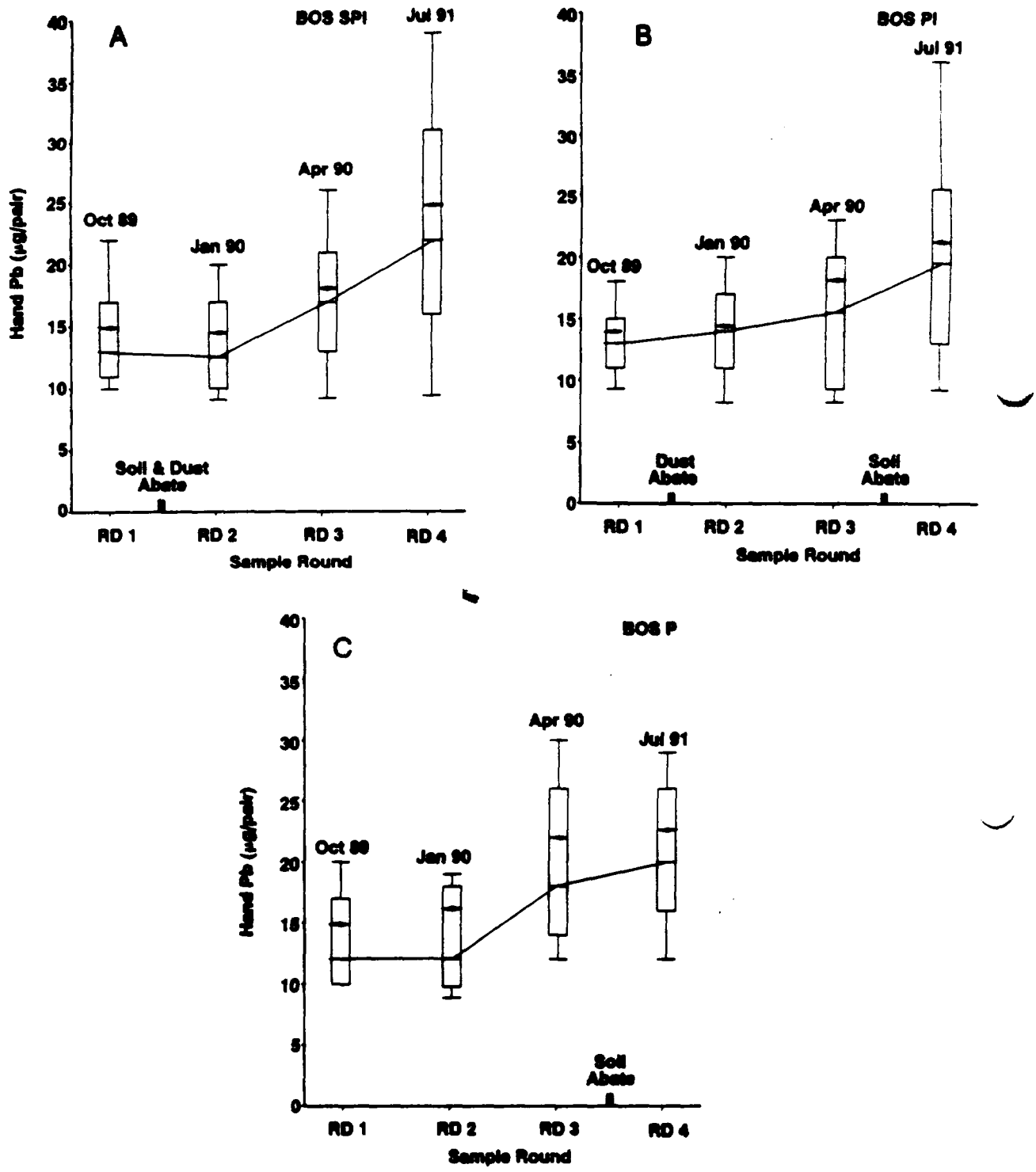

Figure 5-30. Boston hand lead load.

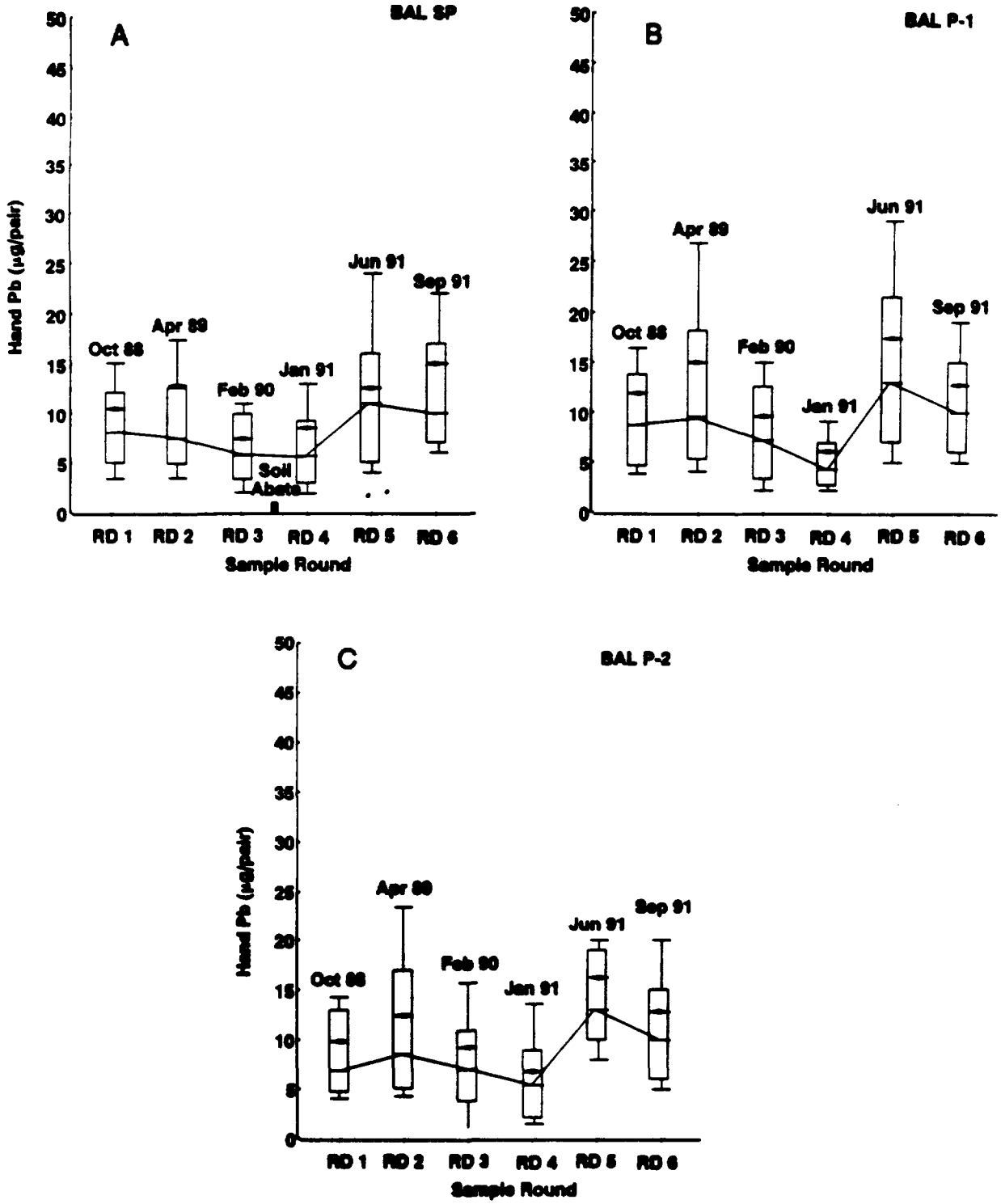

Figure 5-31. Baltimore hand lead load. There were no sequential measurements of Baltimore house dust to compare with the hand lead load.

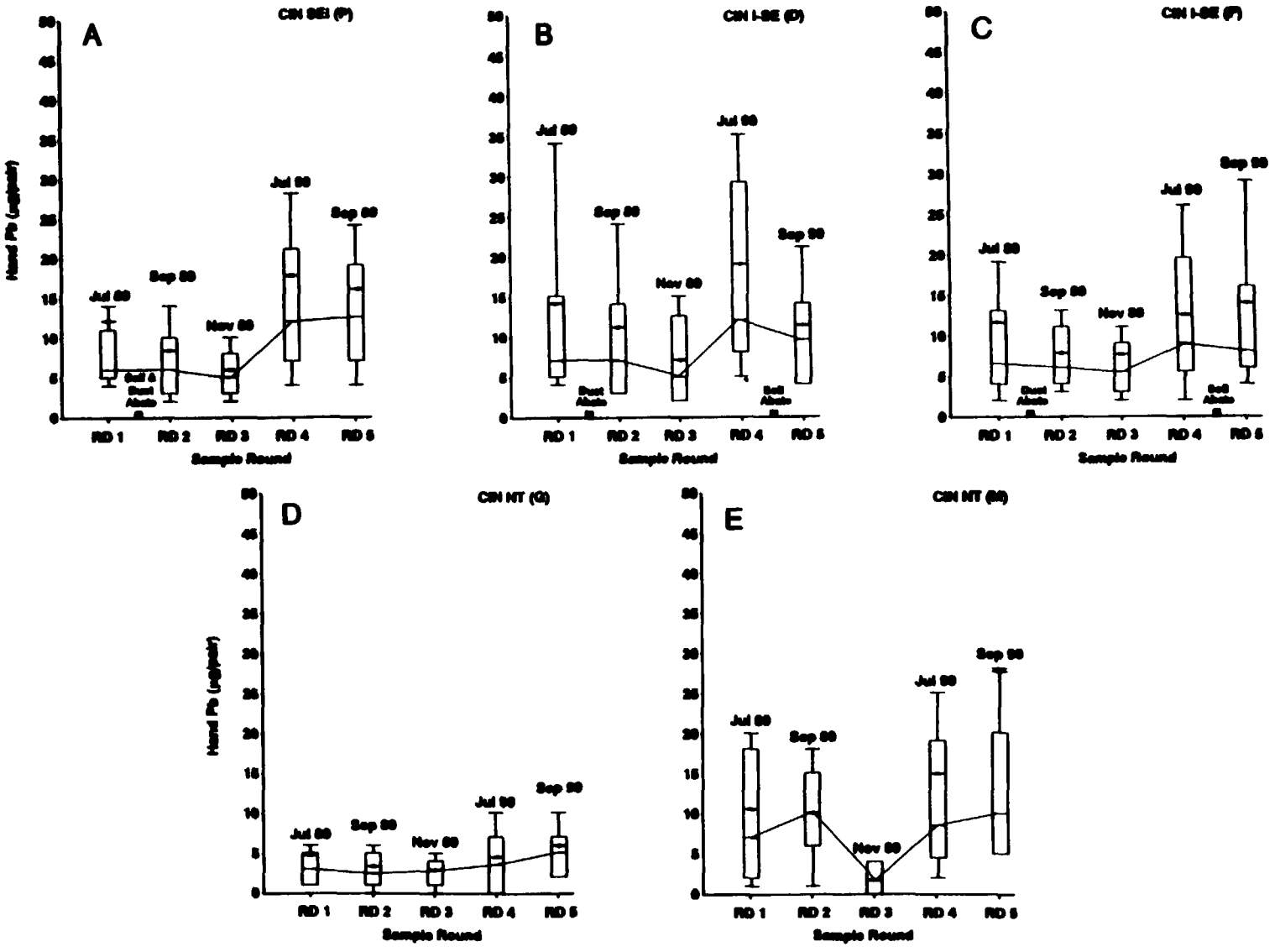

Figure 5-32. Cincinnati hand lead load.

5.2.5 Changes in Blood Lead Concentrations

5.2.5.1 Baltimore Study Blood Lead Data

The blood lead concentrations for the three Baltimore groups are shown in Figure 5-33. The data are for all children participating in the round. They show that the groups were similar prior to soil abatement, but no clear difference between groups in response to intervention. There is a moderate indication of a seasonal cycle comparable to patterns reported in other longitudinal studies, as discussed in Section 2.3.1. The lack of postabatement measurements of soil and house dust limits the ability to interpret these data by more than a simple analysis of variance.

5.2.5.2 Boston Study Blood Lead Data

The blood lead concentrations for the Boston study are shown in Figure 5-34, where they graphically illustrate the conclusions of the Boston report, that intervention probably accounted for a decrease of 0.8 to 1.5 μg/dL in the blood lead.

5.2.5.3 Cincinnati Study Blood Lead Data

The wealth of information from the more detailed measurements of household dust in the Cincinnati study presents a proportionally greater challenge to the modeling of dust exposure pathways. The blood lead concentrations shown in Figure 5-35 show some evidence for seasonal cycles.

5.3 PRE- AND POSTABATEMENT DIFFERENCES IN INDIVIDUALS

5.3.1 Individual Changes in Blood Lead and Soil Lead

Section 5.2 provides a visual presentation of longitudinal patterns in population means for specific parameters over the course of the study. This section presents information on an individual child basis through the use of a series of simple line plots where the blood lead concentrations are plotted by round, age, and study group.

Most children in each neighborhood experienced some change in blood lead, either an increase or decrease, during the course of the study. This change may be due in part to

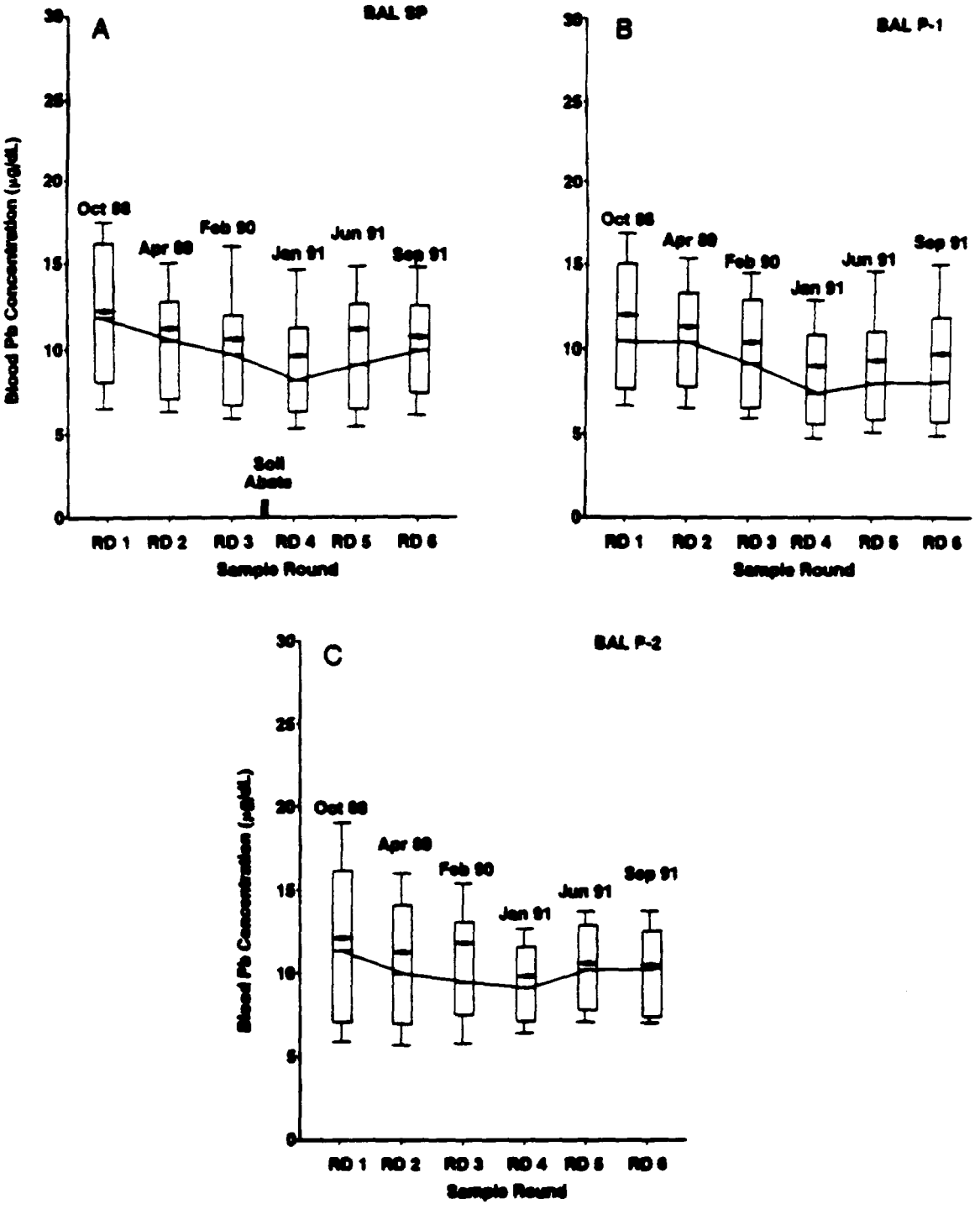

Figure 5-33. Baltimore blood lead concentrations. There appears to be little difference between study groups. Overall, there seems to be a seasonal cycle of the type and magnitude discussed in Section 2.3.1.

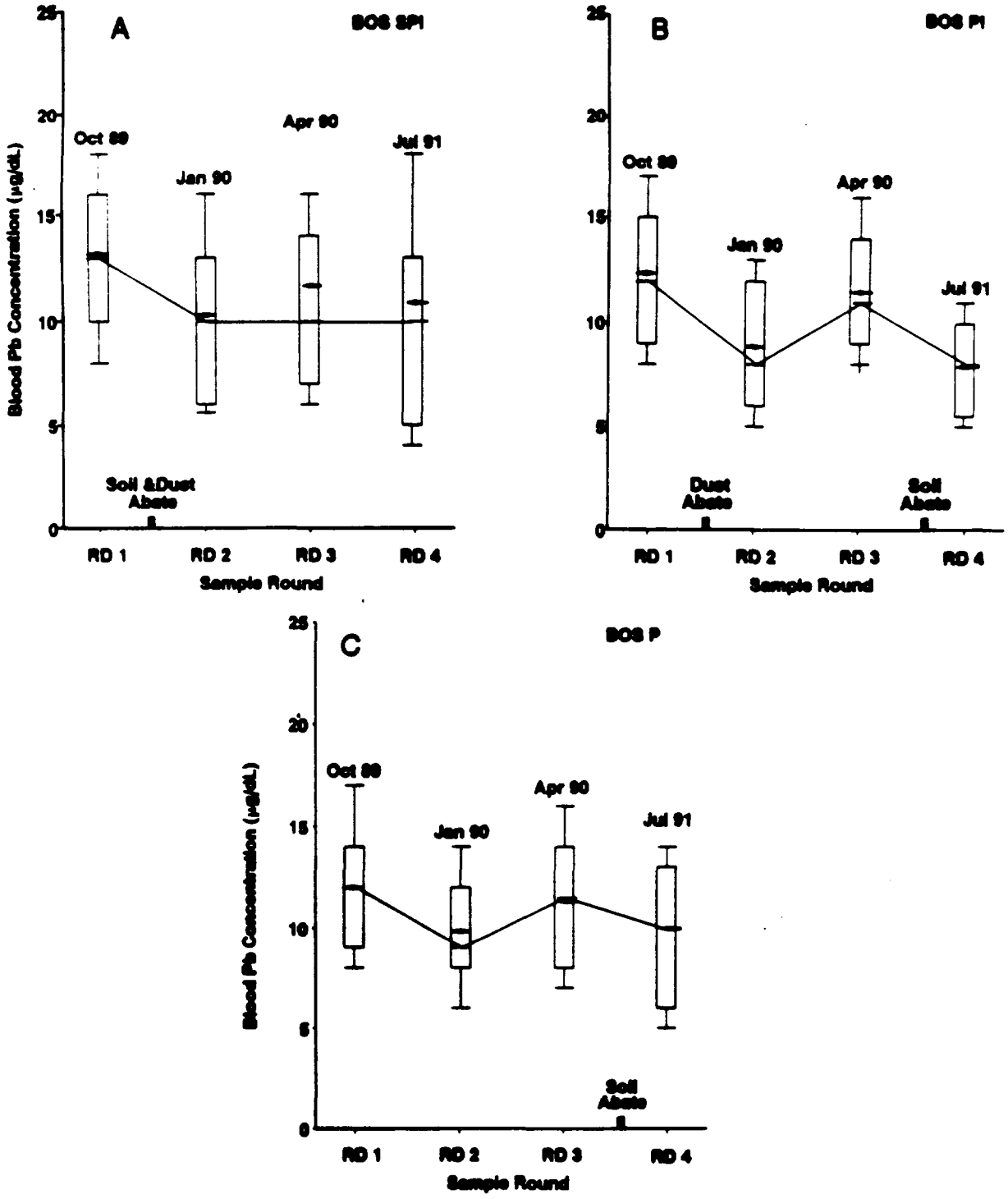

Figure 5-34. Boston blood lead concentrations.

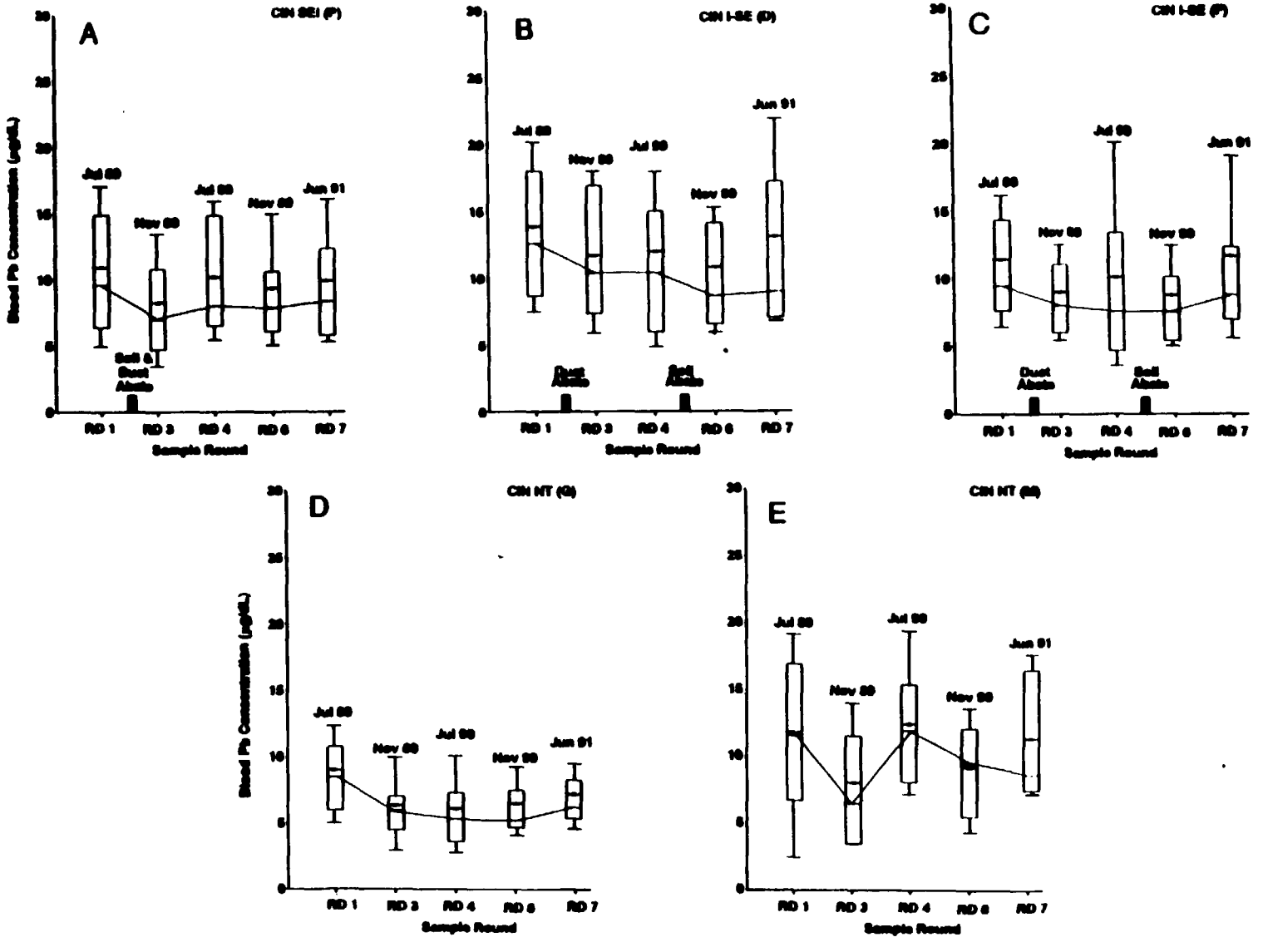

Figure 5-35. Cincinnati blood lead concentrations. Compare to hand lead load patterns in Figure 5-32.

changes brought about by intervention, or to seasonal effects, age (see Figure 2-6), or changes in exposure not related to intervention.

This type of plot is especially helpful to the reader in understanding the variability of the measurements and the possible significance of patterns or clusters. They are designed to show changes in only one variable over time, not the multiple interactions of several variables. In Sections 5.4, 5.5, and 5.6 statistical techniques such as repeated measures analysis and structural equation modeling are used to extract information from the systematic variability using more appropriate methods for comparison than observed on these plots but in the context of several variables interacting at the same time.

Figure 5-36 shows the Boston Phase 1 blood lead observations for each individual child in the three treatment groups broken out by age at the start of the study. Typical patterns show a decrease in blood lead from Round 1 to Round 2 in all groups, including controls, probably attributable in part to seasonal winter decrease. However, in the control group (BOS P-S), there was also a substantial rebound or increase in blood lead at most ages from Round 2 to Round 3, probably attributable to seasonal summer increases. Decreases followed by large increases are noted for most children in the control group BOS P-S, and to a quantitatively greater extent for most children in the Phase 1 dust abatement group BOS PI-S. The Phase 1 soil abatement group BOS SPI shows large decreases between Round 1 and Round 2, similar to the dust abatement group BOS PI-S, but unlike the other groups, many children in BOS SPI show either continuing blood lead decreases between Round 2 and Round 3, or at most very slight summer increases. Five exceptionally large changes are noted. Two children in BOS SPI suffered clear lead poisoning at Round 3, with blood lead increasing to 43 and 35 μg/dL respectively, due to accidental exposure during household renovation. One child in BOS SPI (Round 1 age 18 to 29 months) showed a rather large blood lead increase between Round 2 and Round 3. One child in BOS PI-S (age 18-29 months) showed a large and consistent decrease in blood lead from Round 1 to Round 2 to Round 3, possibly reflecting the effectiveness of interior dust abatement and paint stabilization. One child in P-S (age 42+ months)with blood lead 22 μg/dL also showed a large decrease in blood lead from Round 1 to Round 2 to Round 3, possibly reflecting the role of interior lead paint stabilization as an intervention. We omitted the two children who

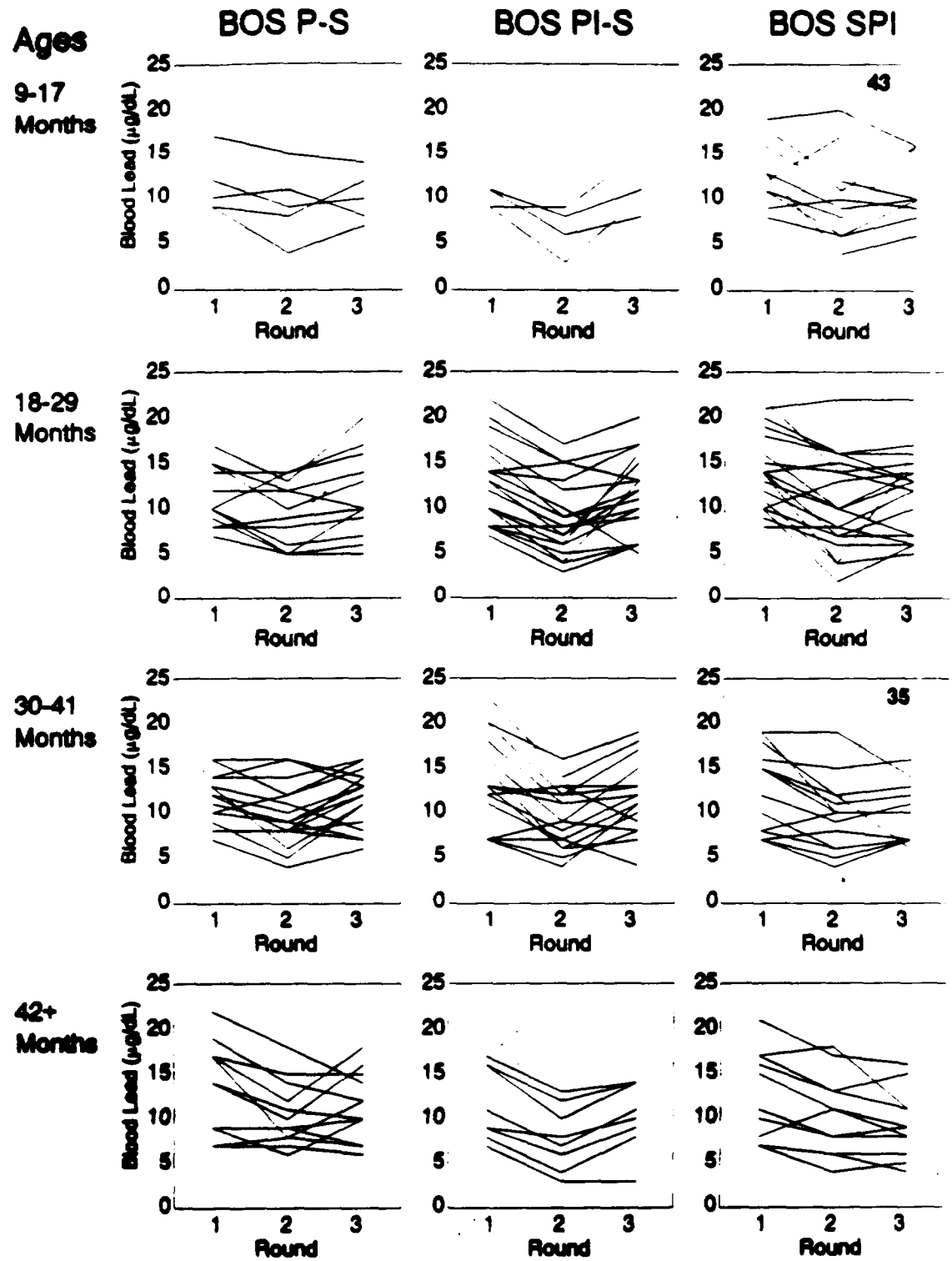

Figure 5-36. Line plots of blood lead for individual children in Phase 1 (rounds 1-3) of the Boston study for each treatment group. Where an individual value exceeds the vertical scale, the actual blood lead concentration is shown near the point of exit, e.g., for two outliers having 35 and 43 µg/dL blood lead concentration.

were lead-poisoned by an identified source from subsequent analyses, but retained all other cases.

Figure 5-37 shows the results of Phase 2 of the Boston study, Rounds 3 to 4. The group BOS PI-S that received dust abatement in Phase 1 and soil abatement in Phase 2 showed a substantial decrease in blood lead for most children; only two children at ages 18 to 29 months showed small increases from Round 3 to Round 4, and one child at age 42+ months showed a relatively large increase. By contrast, 10 children in the group BOS P-S that received only Phase 2 soil abatement showed increases in blood lead during Phase 2, with 2 or 3 increases substantial. By contrast, half of the children in the Phase 2 "control" group BOS SPI, which received Phase 1 soil abatement, showed blood lead increases, 8 of which were substantial increases.

Figure 5-38 shows the Phase 1 results for the Cincinnati study. The three neighborhoods on the left side, CIN I-SE(B) (Back St.), CIN I-SE(D) (Dandridge), and CIN I-SE(F) (Findlay), received interior dust abatement. CIN NT(G) (Glencoe) and CIN NT(M) (Mohawk) received no treatment. CIN SEI(P) (Pendleton), on the right side, received neighborhood soil and street dust abatement as well as interior dust abatement. Many children showed the typical seasonal pattern of higher blood lead in summer (Rounds 1 and 4) and lower in fall or winter (Round 3). Several children in Mohawk (a no-treatment neighborhood) showed large increases, which was much less common in Glencoe, the other no-treatment neighborhood. The other neighborhoods had some children with increased blood lead, and some with decreased blood lead. Many of the increases occurred in children less than 18 months of age, with decreases more evident in children of ages 42+ months. There was not any external basis for omitting children from subsequent analyses.

Figure 5-39 shows the Phase 2 results for Cincinnati. There is even less pattern for Phase 2 than for Phase 1, as was subsequently verified by detailed analyses.

Figure 5-40 shows the results for the Baltimore study. Soil abatement was carried out in group BAL SP a few months after Round 3. While there were substantial decreases in blood lead in some children in group BAL SP after Round 3, there were many who did not show any decrease. Two children in BAL SP (ages 18-29 and 54-65 months) showed very large increases between Rounds 3 and 4, and one child age 30-41 months showed a large increase between Round 4 and Round 5. A number of children in the Area 2 control group

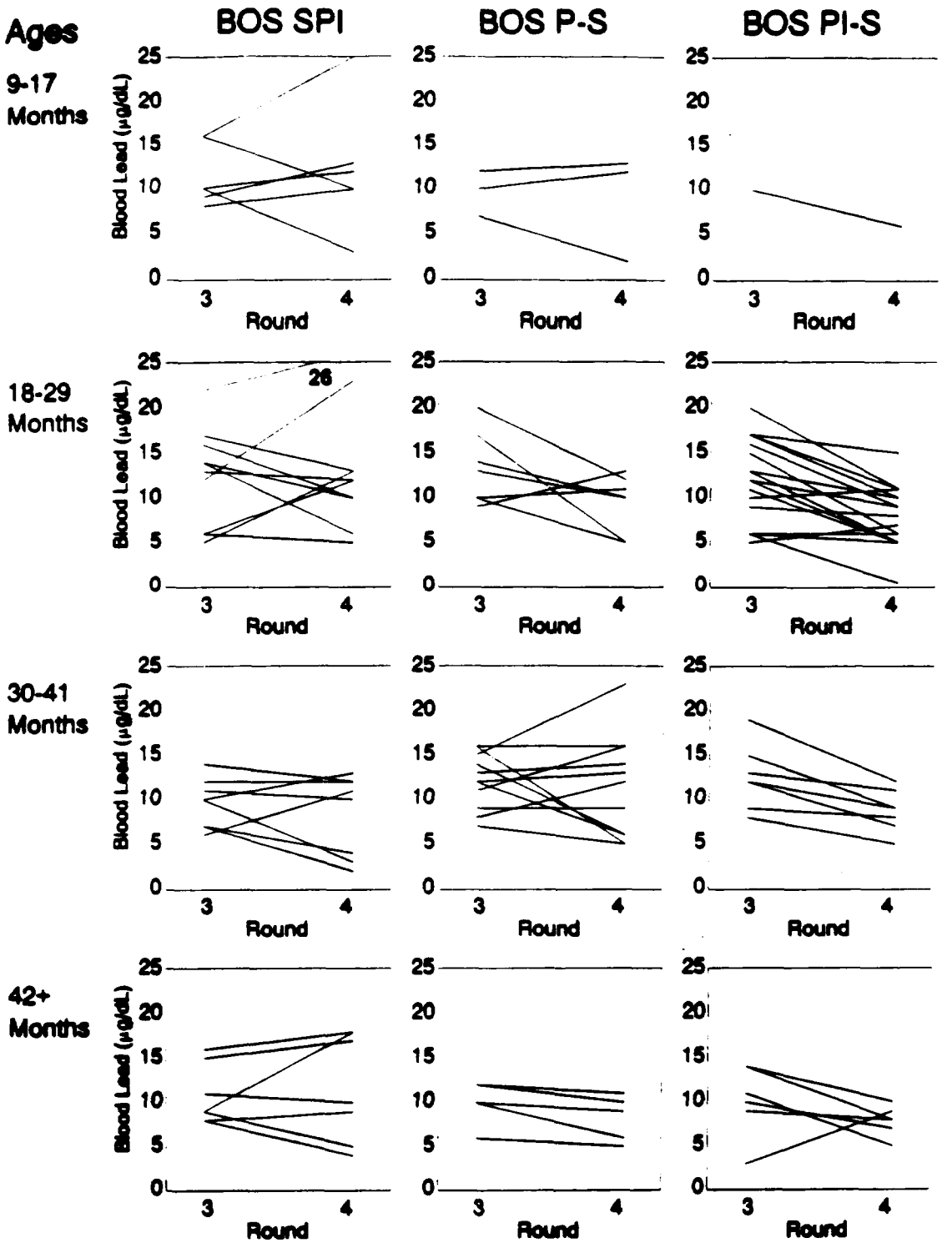

Figure 5-37. Line plots of blood lead for individual children in Phase 2 (Rounds 3 and 4) of the Boston study for each treatment group.

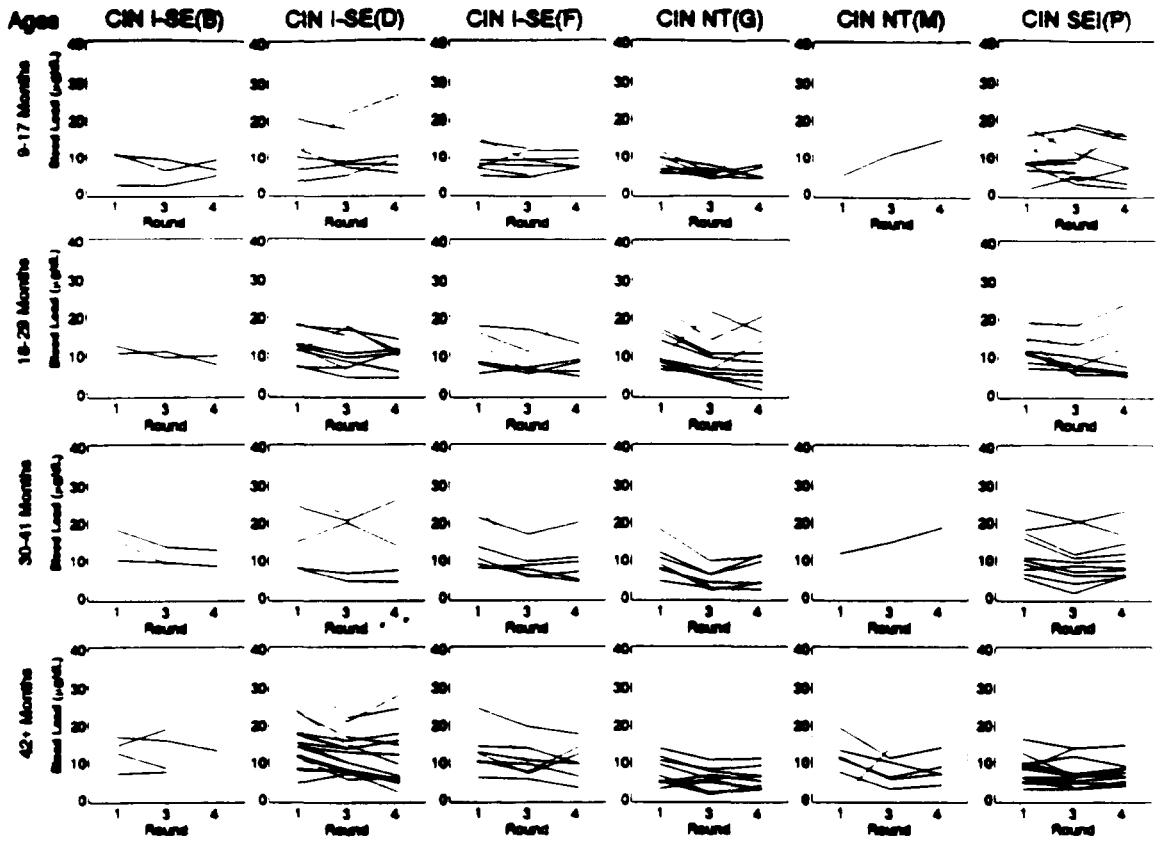

Figure 5-38. Line plots of blood lead for individual children in Phase 1 (Rounds 1-4) of Cincinnati for each treatment group and four age groups.

(BAL P1) showed large increases, particularly between Round 4 and Round 6, but many also showed large decreases in blood lead after Round 3, particularly at ages 18-29, 54-65, and 66+ months. The Area 1 control group BAL P2 showed no dramatic changes in blood lead. Note that the Baltimore cohort was typically much older at Round 3, the last pre-abatement round. While there were a few children less than 18 months in the study, they were mostly in group BAL SP and are not shown in Figure 5-40. No children were omitted from subsequent analyses based on external evidence.

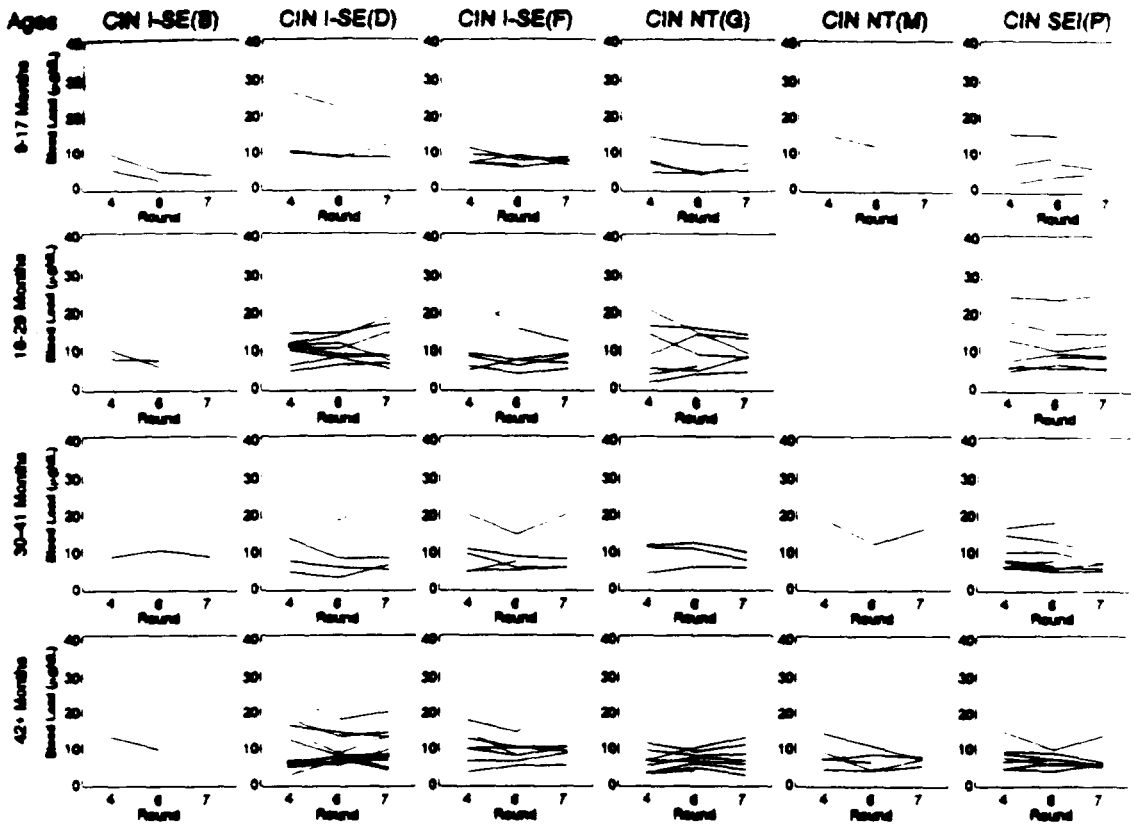

Figure 5-39. Line plots of blood lead for individual children in Phase 2 (Rounds 4-7) of Cincinnati for each treatment group and four age groups.

5.4 COMPARISON BY CROSS-SECTIONAL STRUCTURAL EQUATION MODELS

The cross-sectional structural equation model (XSEM) is a useful tool for answering the question of whether a source of lead such as soil is a major component of environmental lead exposure for the children. Lead in household dust, particularly floor dust, is often the most important source of lead exposure for children, but house dust is not generally regarded as a primary source because the lead in house dust is usually derived from other sources. The sources of lead in house dust include lead in soil that is tracked into the house, lead from deteriorating lead-based paint, lead dust from occupational exposure that is carried into the house on shoes and clothing, lead particles deposited from the air (including resuspended

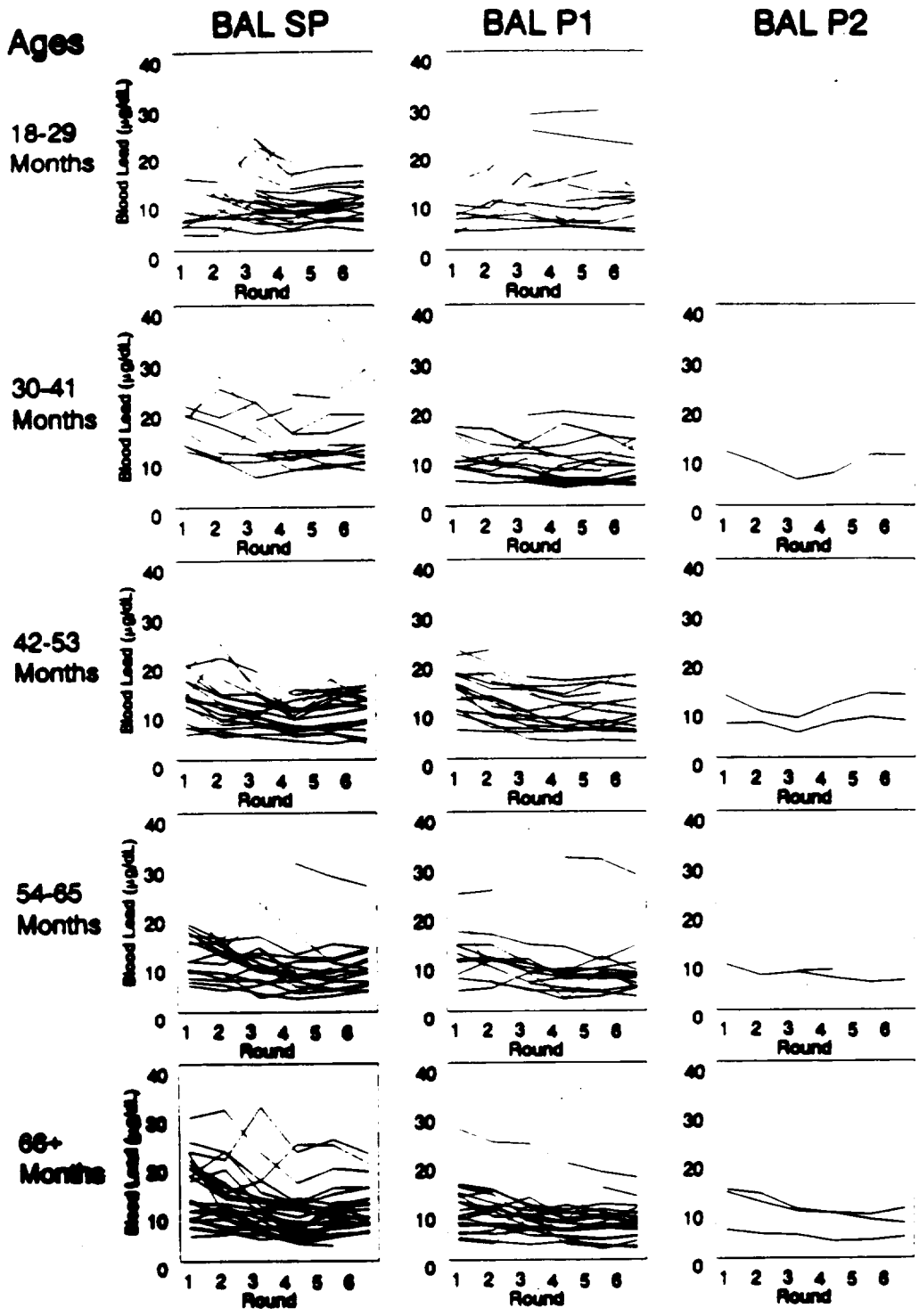

Figure 5-40. Line plots of blood lead for individual children in Baltimore for each treatment group and five age groups.

surface soil particles), and from household activities such as lead hobbies or home occupations. Soil lead may therefore contribute to blood lead as both a direct exposure source during outdoor activity, and indirectly as a source of lead in household dust.

5.4.1 General Issues in Structural Equation Modeling

The general conceptual model shown in Figure 5-41, based on Figure 2-3, is the basis for the structural equation models fitted in this chapter. None of the three studies measured airborne lead concentrations, which generally provide only small additions to child lead exposure beyond the soil lead and dust lead particles historically deposited from leaded gasoline, industrial emissions, and other sources of airborne particles. Fugitive emissions from deleading or demolition of older structures or from soil excavation may have occurred, but were not systematically observed.

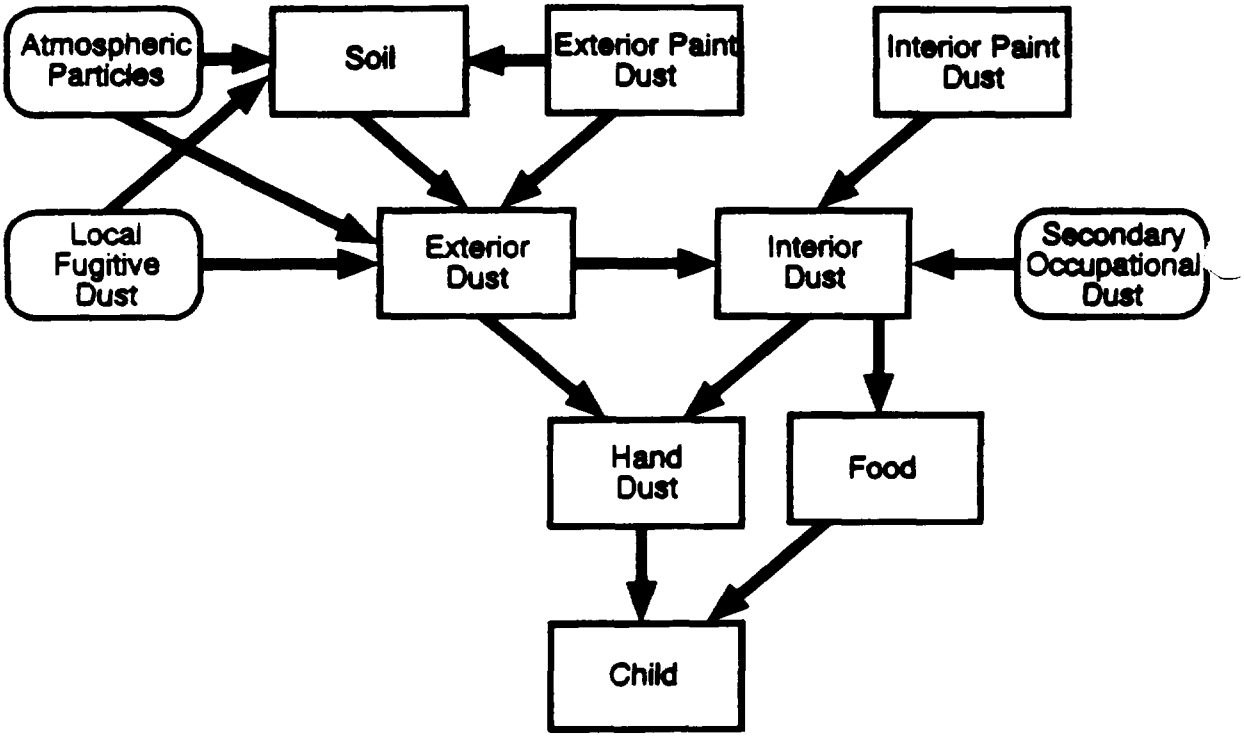

Figure 5-41. Typical pathways of childhood exposure to lead in dust showing both the complexity of the routes of exposure and the mobility of dust lead along these routes.

The general graphic representation for both cross-sectional and longitudinal structural equation models is shown in Figure 5-42. The variables in the system can be classified as *independent variables* or *dependent variables*. Independent variables are not predicted by any other variables in the system, and may vary by neighborhood, treatment group, household, residence, or child. They are shown by oval figures with no arrows going into them, and are also known as predictor variables or exogenous variables. Dependent variables are shown as rectangular figures with at least one arrow going into them, and are also known as response variables or endogenous variables. Dependent variables require input from at least one other component of the system, either from independent variables or other dependent variables. The arrow shows the direction of the relationship, with the dependent variable being predicted by a multiple linear or nonlinear regression model using the variables at the initial part of the arrow. Parameters whose estimates are shown in the tables of this chapter are either regression coefficients in the equations, or intercept terms in the equations from Section 5.1.2.

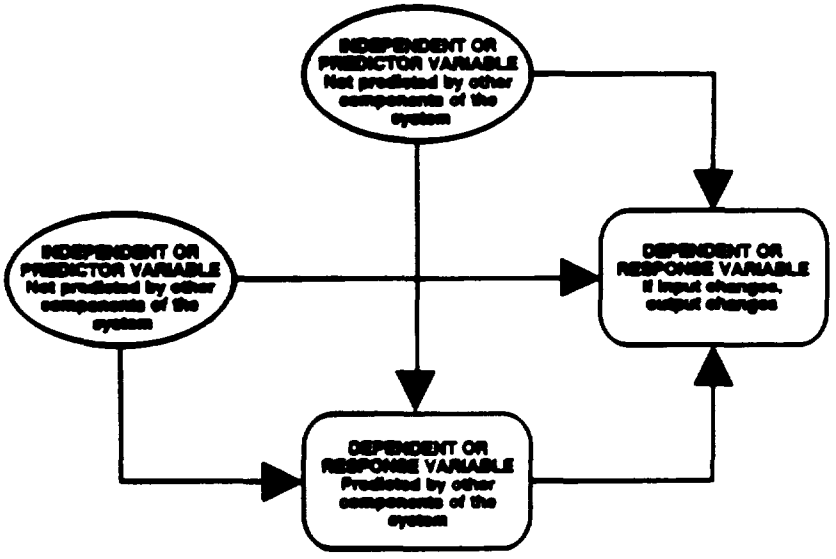

Figure 5-42. Explanation of the terms and features of the structural equation model.

Figure 5-43 shows an expanded version of Figure 5-41, with greater emphasis on the variables used for structural equation modeling. Exterior and interior lead paint dust was not measured, but data are available on lead paint loadings on walls or trim. However, any relationship between lead paint and house dust was compromised in all of the studies, either by interior paint stabilization in Boston, by exterior paint stabilization in Baltimore, or by restricting the study to fully rehabilitated houses primarily in Cincinnati. Window dust lead proved to be a significant predictor of floor dust lead that was distinguishable from lead in soil in Boston and Cincinnati, but somewhat less predictive of blood lead, and so occupies a position similar to soil in the pathway scheme. Floor dust lead was strongly correlated with post-abatement blood lead and is shown as a more proximate predictor of blood lead in the pathway models.

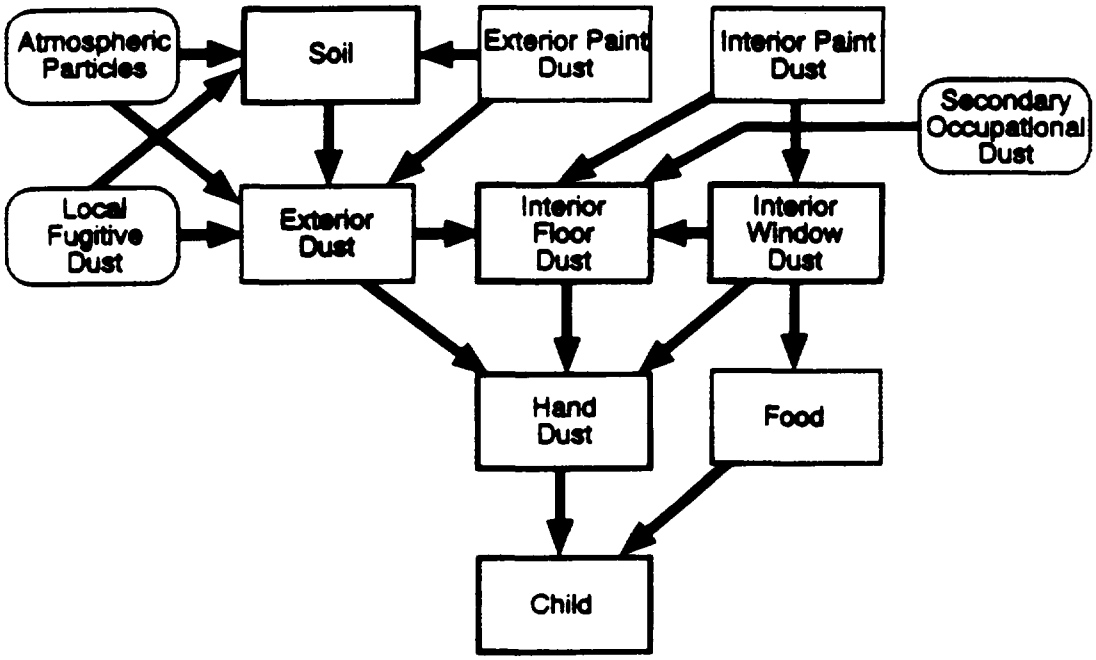

Figure 5-43. Dust lead exposure pathway diagram, similar to Figure 5-41, showing the assumed relationships for interior floor and window dust that were modeled by cross-sectional SEM. Shaded compartments were not included in the XSEM analyses.

The main focus of the structural equation analyses is to evaluate the predictive role of environmental lead measurements in dust and soil, rather than child-specific measurements in which the child is used as a sentinel of changes in exposure. Therefore, we used a further simplification of Figure 5-43 to develop the final structural equation model shown in Figure 5-44 that was used as basis for the analyses reported for Boston and Cincinnati. Soil lead and window dust lead were used as independent predictors of both floor dust lead and blood lead. Window dust lead was used to characterize non-soil exterior exposures and interior exposures not characterized by floor dust lead. Hand dust lead was omitted as a predictor of blood lead, replaced by the floor dust lead, window dust lead, and soil lead as direct predictors.

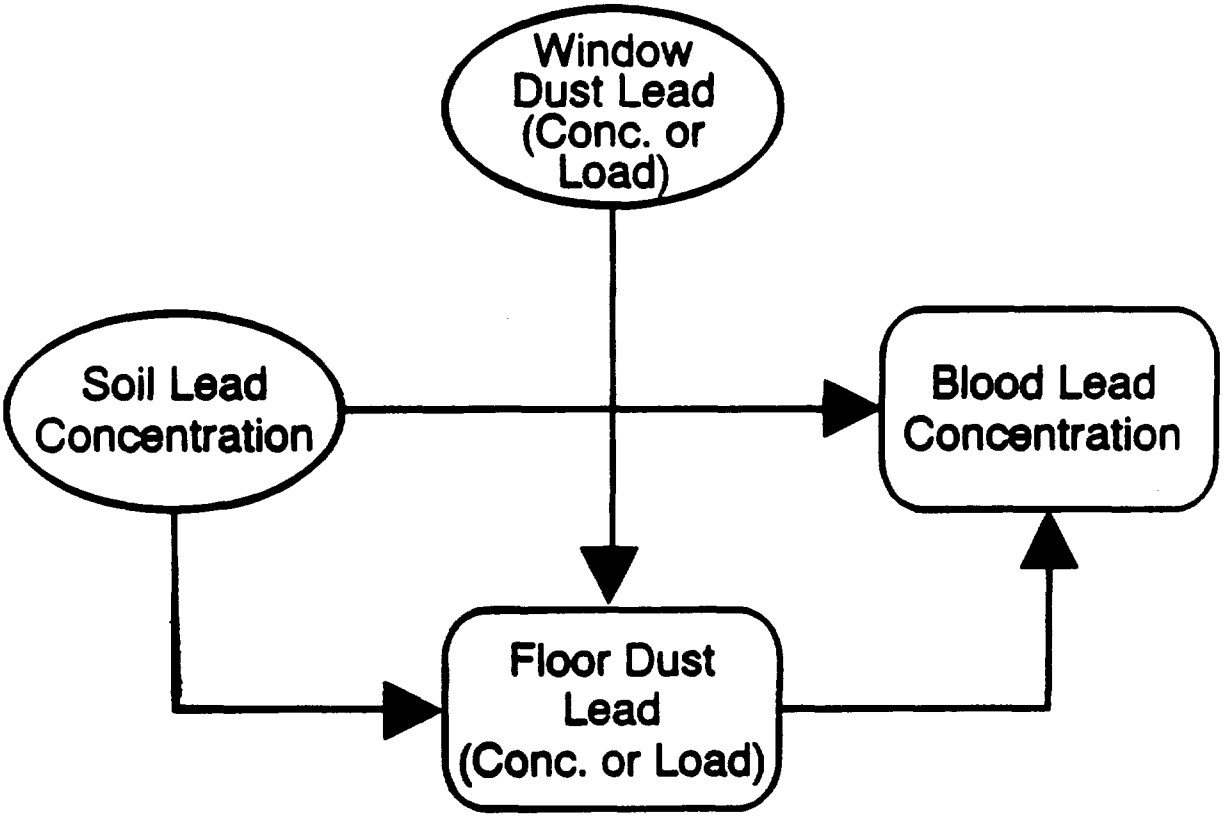

Figure 5-44. Adaptation of the soil and dust pathway diagram (Figure 5-43) that illustrates the general scheme for the cross-sectional structural equation models, using the notation of Figure 5-42.

More complex models involving additional dependent or independent variables or pathways would greatly increase the complexity of the analyses, especially when the cross-sectional models are linked longitudinally so as to assess effects over time. The simple models presented here already include a large number of parameters that need to be estimated from the data. With increasing complexity, the iterative estimation procedures used to fit the structual equations fail to converge to a unique optimal solution for the parameter estimates, and such complexity should be added only if there is a compelling substantive scientific basis for doing so. In most applications, adding additional parameters and interaction terms for age effects and gender differences was less informative than simply stratifying the data by age or gender.

We evaluated a number of two-equation models in which the first equation represented blood lead concentrations in children derived from environmental pathways and media including soil and floor dust, and the second equation represented floor dust lead concentration or lead loading derived from lead in exterior media, including soil lead and lead on window sills or in window wells.

Blood lead concentrations are related to lead in soil and to lead loading or concentration in house dust at or shortly before blood leads are measured, as well as to prior or historic lead exposures that have accumulated a (primarily skeletal) body burden of lead that contributes to current blood lead concentrations. The child's age as well as many other individual behavioral or demographic factors may also affect exposure. Although it is not necessary to dwell on the concept that there is a "causal" implication for any proposed predictive relationship, it should be noted that in a longitudinal lead study, some of the lead in the child's body (even in blood and soft tissues) will be circulating in blood at a later measurement. Thus, estimates of blood lead concentrations in earlier samples are expected to be predictive of measurements from later samples, which are estimates of the same quantity, in part. The models do not depend on causal interpretations, however, but do assume a temporal direction in which the dependent variables depend on values of other variables measured at the same time, or measured previously, but not on values measured in the future.

Structural Equation Modeling is a computational approach that allows estimation of sets of inter-related linear or nonlinear models (Buncher et al., 1991). This has been widely used

for cross-sectional environmental pathway modeling (Bornschein et al., 1985, 1988, 1990; Marcus, 1991, 1992). Applications to longitudinal lead studies have recently been developed (Marcus, 1991; Menton et al., 1994; Marcus and Elias, 1994). The PROC MODEL program in the SAS ETS computer package (SAS, 1992) allows estimation of either linear or nonlinear models. This procedure is believed to produce less biased estimates of regression coefficients than other estimation procedures that do not include fitting simultaneous equations for blood lead to predictor variables such as lead in paint, soil, or dust.

The most complete and technically correct evaluation of the present three studies requires simultaneous assessment of changes in blood lead levels and changes in environmental lead pathways following soil lead or dust lead abatement. The underlying assumptions in the Structural Equation Model approach are that abatement effects can be inferred from changes in environmental lead exposure variables. Because this is a cause-effect relationship, it is sequence-dependent or time-dependent. That is, the abatement must take place before the environmental changes will occur. The cross-sectional SEM models use correlation structure in the data to infer causal pathway relationships. The longitudinal SEM models also use correlation structure to infer causality, but the logical basis for inference is much stronger because the interventions or abatements precede the changes in blood lead and environmental lead. Changes in control groups during the same period of time then provide a basis for estimating treatment effects. Any analysis of time-dependent relationships should address the following assumptions:

(1) Both preabatement and postabatement blood lead levels reflect, in part, contemporary environmental lead exposures that can be characterized by measurements of lead levels in soil, dust, paint, and other media;

(2) Postabatement blood lead levels may also reflect, in part, preabatement blood lead levels due to the contribution of preabatement body burdens of lead (principally in the skeleton) from earlier exposures;

These models were fitted using indicator or "dummy" variables for different study or treatment groups. Sometimes these indicator variables were used as "switches", for example when postabatement soil lead concentration is modeled as a fraction of preabatement soil lead for soil nonabatement groups, but as a new replacement value for the soil abatement groups. At other times, indicator variables were used when the data suggested that the effect of abatement was to modify the regression coefficient for the predicted variable (for example,

floor dust lead concentration) for a pathway. In that case, separate coefficients were fitted to the product of the treatment group indicator and the predictor variable (for example, entry dust lead concentration) as well as separate intercept terms for each treatment group.

The purpose of structural equation modeling is to elucidate pathways for environmental lead exposure from source to child. From this perspective, the development and testing of pathway models for urban lead is an exploratory model-building activity that does not readily lend itself to hypothesis testing. It is well known that "specification searches" such as step-wise regression have complicated inferential properties (Leamer, 1978), and the true P level for an estimated regression coefficient may be quite different from the nominal P value. An up-and-down search procedure was employed that started with a plausible pathway diagram, and dropped nonsignificant blocks of parameters if all estimates of the same or analogous parameters in different groups were zero or nonsignificant. New parameters were added for each new pathway in the model, based on prior beliefs and on sample correlation coefficients.

5.4.2 Boston Preabatement Cross-sectional Structural Equation Models

The model scheme for the Boston cross-sectional structural equation modeling is shown in Figure 5-45, using the notation of Figure 5-44. The results for the twelve Boston models with dust lead concentration are shown in Table 5-1, and an example of Model 1 with the output parameters is shown in Figure 5-46. The cross-sectional structural equation model coefficients in these four tables correspond to equations 5-7 and 5-8 of Section 5.1.2, which are repeated here for convenience.

$$Y_{ir} = G_{gr} + X_{ir}B_{gr} + Z_{ir}F_{gr} + e_{ir} \qquad (5\text{-}7)$$

The blood lead models in Equation 5-7 have a single intercept term (denoted G) for each round. The dust-to-blood regression coefficient (denoted B) is usually assumed to be the same for all groups. The soil-to-blood lead regression coefficient (denoted F) is also shown. The dust lead models follow the form of Equation 5-8, where the floor dust lead

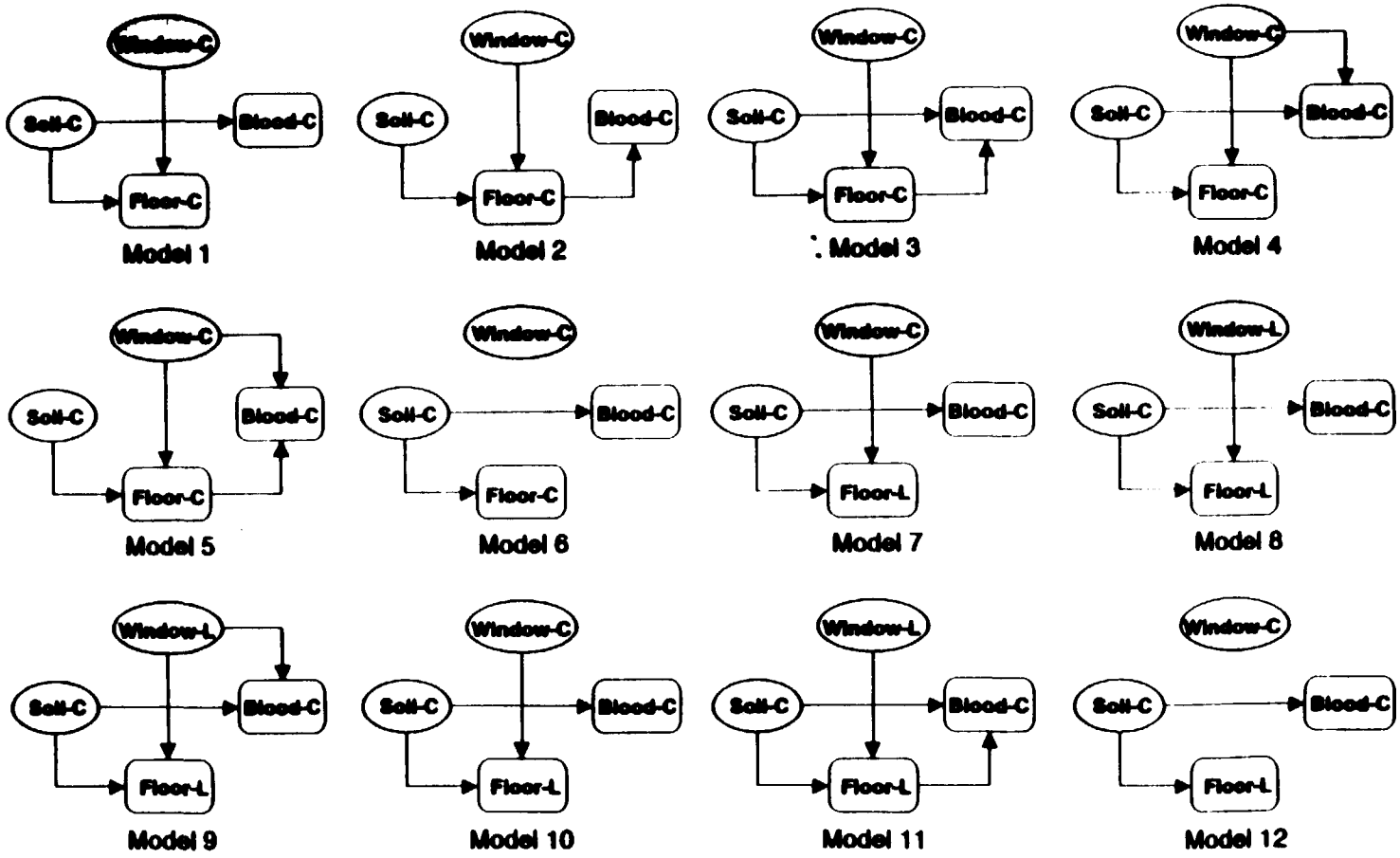

Figure 5-45. Pathway diagram of twelve different cross-sectional structural equation models for Round 1 of the Boston study. Arrows show regression models for blood lead and floor dust lead concentrations. Oval figures show soil lead

TABLE 5-1. PREABATEMENT CROSS-SECTIONAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY

	SEM EQUATION COEFFICIENTS		FLOOR DUST LEAD CONCENTRATION					
			Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	INTERCEPT[1]	G_{gr}	11.58[S4]	11.56[S4]	10.97[S4]	11.20[S4]	11.92[S4]	10.83[S4]
SLOPE	Floor → Blood[2]	B_{gr}		0.11	0.14		-0.36[S2]	
SLOPE	Soil → Blood[2]	F_{gr}	0.13		0.16	0.12		0.32[~]
SLOPE	Window → Blood[2]	F_{gr}				0.0182	0.0694[~]	
	INTERCEPT[3]	C_{gr}	986[S2]	961[S2]	1008[S2]	1014[S2]	993[S2]	1347[S3]
SLOPE	Soil → Floor[4]	D_{gr}	0.074	0.090	0.075	0.076	0.081	0.331[S1]
SLOPE	Window → Floor[5]	D_{gr}	0.0657[S4]	0.0652[S4]	0.0651[S4]	0.0647[S4]	0.0644[S4]	
			FLOOR DUST LEAD LOADING					
	INTERCEPT[1]	G_{gr}	11.37[S4]	11.34[S3]	11.23[S4]	11.00[S4]	10.96[S4]	11.13[S4]
SLOPE	Floor → Blood[6]	B_{gr}					2.74	
SLOPE	Soil → Blood[2]	F_{gr}	0.18	0.20	0.19	0.17	0.19	0.27[~]
SLOPE	Window → Blood[2] dust Pb conc	F_{gr}			0.0073			
SLOPE	Window → Blood[6] dust Pb load	F_{gr}				0.0191		
	INTERCEPT[3]	C_{gr}	12.0	28.3[S1]	28.9[S1]	11.7	28.9[S1]	47.8[S2]
SLOPE	Soil → Dust[7]	D_{gr}	0.0033	0.0024	0.0022	0.0036	0.0023	0.0147[S1]
SLOPE	Window → Floor[7] dust Pb conc	D_{gr}	0.0031[S4]			0.0031[S4]		
SLOPE	Window → Floor[8] dust Pb load	D_{gr}		0.0048[S2]	0.0046[S3]		0.0046[S3]	

Note: In this and all subsequent tables of this chapter, the following notation is used to indicate statistical significance:
S4 - Significance Level 4, P value < = 0.0001.
S3 - Significance Level 3, P value 0.0002 - 0.0019.
S2 - Significance Level 2, P value 0.002 - 0.0099.
S1 - Significance Level 1, P value 0.01 - 0.0499.
1T - Nearly significant or one-tailed significance, P value 0.05 - 0.0999.
M - Marginally significant, P value 0.1 - 0.1999.

[1]Units are μg/dL Pb in blood.
[2]Units are μg/dL Pb in blood per 1000 μg/g Pb in soil.
[3]Units are μg/g Pb in dust.
[4]Units are μg/g Pb in dust per μg/g in soil.
[5]Units are μg/g Pb in dust per μg/g in dust.
[6]Units are μg/dL Pb in blood per 1000 μg/m² in dust Pb load.
[7]Units are μg/m² Pb in dust per μg/g Pb in soil.
[8]Units are μg/m² Pb in dust per μg/m² Pb in dust.

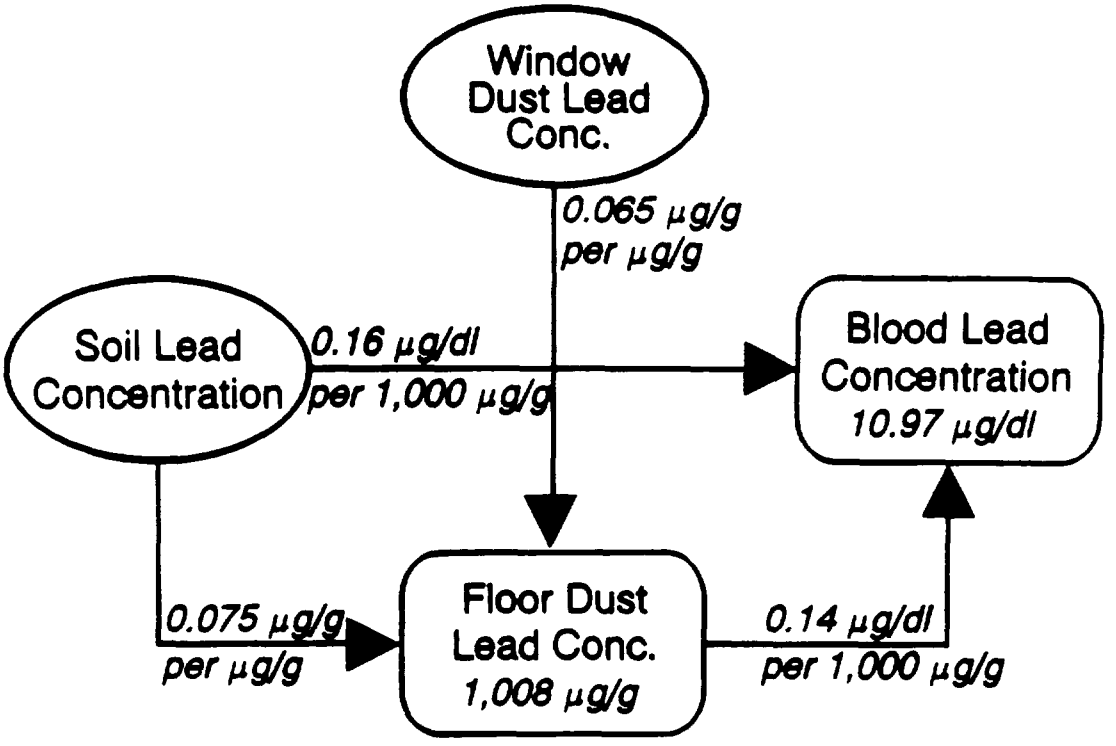

Figure 5-46. Pathway diagram for Boston cross-sectional SEM Model 3, with results as indicated from Table 5-1.

models have a single intercept term (denoted C) and a single soil-to-dust regression coefficient (denoted D).

In general, any of several models gave an almost equally good fit to the preabatement blood lead and dust lead data. However, none of the blood models provided a significantly better prediction than did the group mean. The most significant finding ($P=0.12$) was for Model 6 in Table 5-1, with soil lead concentration as the only preabatement blood lead predictor. However, this model also used soil lead as the predictor of dust lead concentration, and provided a significantly worse prediction of dust lead than did any of the models that used window lead as well as soil lead as predictor of floor dust lead. In fact, preabatement soil lead was never a significant predictor of dust lead when window lead was included in the model. This indicates that, at the Boston preabatement stage, a substantial amount of the lead in floor dust may have come from the window dust, perhaps as lead-based paint, rather than the soil. Very similar results were found when floor dust lead

loading was used as the dust index, as shown also in Table 5-1, and the analogous blood lead-soil lead coefficient was even less significant (P = 0.18).

Because the Boston study excluded children whose blood lead concentration might be too low (< 7 μg/dL) to be accurately measured following an expected reduction of three or more μg/dL, or so high (> 24 μg/dL) that the child would require immediate medical intervention, there was a concern that this truncation at both ends might have biased the statistical analyses. To address this concern, the Boston data set was artificially truncated to the interval 9-22 μg/dL, and a number of marginally significant relationships emerged. Table 5-2 shows that the soil lead concentration by itself, or in combination with the floor dust lead concentration or window dust lead lead concentration, provides a marginally significant preabatement predictor of blood lead. The dust lead variables have positive coefficients in models with soil lead, and are marginally significant predictors by themselves. Due to the collinearity between floor dust lead and window dust lead, neither is significant when used together in a model with soil lead, and while both were more significant when used together in a model without soil lead, the floor dust coefficient was negative. Similar results were obtained in the model for floor dust lead loading with blood lead truncation shown in Table 5-2.

5.4.3 Cincinnati Cross-Sectional Structural Equations Model

The Cincinnati study collected dust lead measurements at a number of locations. One of the goals of the Cincinnati cross-sectional structural equations modelling exercise was to evaluate the ability of different dust lead indices to predict blood lead and, in turn, the relationship of that dust lead index to soil lead. Four different dust lead locations were considered, using both lead concentration and lead loading. The locations were: composite interior floor, interior entry, and window sill. The models evaluated were very simple, with soil lead-dust lead-blood lead and soil lead-blood lead pathways. Results are shown in Table 5-3.

The most useful models for predicting blood lead at Round 1 used floor dust or entry dust lead concentration and loading, but none of the regression coefficients for blood lead versus dust lead were statistically significant. In the model in which both floor dust and soil lead concentration were used, the blood lead versus floor dust lead coefficient was small

TABLE 5-2. PREABATEMENT CROSS-SECTIONAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: BLOOD LEAD TRUNCATED (9-22 μg/dL)

	SEM EQUATION COEFFICIENTS		FLOOR DUST LEAD CONCENTRATION					
			Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	INTERCEPT[1]	G_{gr}	12.43[S3]	11.51[S4]	11.90[S4]	11.77[S4]	12.99[S4]	12.34[S4]
SLOPE	Floor → Blood[2]	B_{gr}		0.205[M]		0.063	-0.377[S1]	
SLOPE	Soil → Blood[2]	F_{gr}	0.268[M]	0.305[M]	0.250[M]	0.267		0.328[M]
SLOPE	Window → Blood[2]	F_{gr}			0.0221[M]	0.0151	0.0788[1T]	
	INTERCEPT[3]	C_{gr}	770[S1]	750[S1]	755[S1]	753[S1]	821[S1]	1544[S2]
SLOPE	Soil → Floor[4]	D_{gr}	0.106	0.113	0.111	0.112	0.089	0.335[S1]
SLOPE	Window → Floor[5]	L_{gr}	0.0760[S4]	0.0750[S4]	0.0752[S4]	0.0751[S4]	0.0765[S4]	
			FLOOR DUST LEAD LOADING					
			Model 7	Model 8	Model 9	Model 10	Model 11	Model 12
	INTERCEPT[1]	G_{gr}	12.40[S4]	12.46[S4]	11.76[S4]	11.83[S4]	12.23[S4]	12.24[S4]
SLOPE	Floor → Blood[6]	B_{gr}			3.94			
SLOPE	Soil → Blood[2]	F_{gr}	0.264[M]	0.250	0.276[M]	0.251[M]	0.263[M]	0.358[1T]
SLOPE	Window → Blood[2] dust Pb conc	F_{gr}				0.025[M]		
SLOPE	Window → Blood[6] dust Pb load	F_{gr}					0.083	
	INTERCEPT[3]	C_{gr}	91.7	30.7[S1]	30.7[S1]	9.2	30.7[S1]	51.7[S2]
SLOPE	Soil → Dust[7]	D_{gr}	0.0043	0.0019	0.0015	0.0041	0.0014	0.0139[S1]
SLOPE	Window → Floor[7] dust Pb conc	L_{gr}	0.0031[S4]			0.0031[S4]		
SLOPE	Window → Floor[8] dust Pb load	L_{gr}		0.0025[S2]	0.0046		0.0046[S2]	

[1]Units are μg/dL Pb in blood.
[2]Units are μg/dL Pb in blood per 1000 μg/g Pb in soil.
[3]Units are μg/g Pb in dust.
[4]Units are μg/g Pb in dust per μg/g in soil.
[5]Units are μg/g Pb in dust per μg/g in dust.
[6]Units are μg/dL Pb in blood per 1000 μg/m^2 in dust Pb load.
[7]Units are μg/m^2 Pb in dust per μg/g Pb in soil.
[8]Units are μg/m^2 Pb in dust per μg/m^2 Pb in dust.

TABLE 5-3. PREABATEMENT CROSS-SECTIONAL STRUCTURAL EQUATION MODELS FOR CINCINNATI STUDY: DUST TYPE MODELS

SEM EQUATION COEFFICIENTS		With No Soil → Blood Slope			With Soil → Blood Slope			
		Floor	Entry	Window	Floor	Entry	Window	Soil
DUST LEAD CONCENTRATION ALL AGES[1,2]								
INTERCEPT[1]	G_g	7.41[S4]	7.02[S4]	6.73[S2]	8.47[S3]	2.52[M]	9.10	8.47[S4]
Slope: Dust → Blood[2]	B	4.65	5.35	1.11	-0.02	26.4[S4]	-0.224	
Slope: Soil → Blood[2]	F				1.72	-4.08[1T]	2.54	1.72[S1]
INTERCEPT[3]	C_g	160[S4]	182[S4]	908[S4]	160[S4]	185[S4]	908[S4]	160[S4]
Slope: Soil → Dust[4]	D	0.330[S3]	0.317[S3]	1.24[S2]	0.331[S3]	0.310[S3]	1.24[S2]	0.33[S3]
DUST LEAD LOADING ALL AGES								
INTERCEPT[1]	G_g	8.80[S4]	8.69[S4]	8.82[S4]	8.65[S4]	8.57[S4]	8.45[S4]	8.54[S4]
Slope: Dust → Blood[6]	B	0.93	0.23	0.0087	-0.25	0.10	-0.001	
Slope: Soil → Blood[2]	F				2.18	0.823	1.98	1.58
INTERCEPT[3]	C_g	15.5	34.6	1402[1T]	15.0	36.0	1321[S4]	15.0
Slope: Soil → Dust[7]	D	0.146[S2]	0.216[S1]	0.458	0.149[S2]	0.210[S1]	0.654	0.149[S2]
DUST LEAD CONCENTRATION for AGE 42+ MONTHS[1,2]								
INTERCEPT[1]	G_g	-1.02	3.16	1.58	8.43[S4]	8.30[S3]	8.13[S4]	6.95[S4]
Slope: Dust → Blood[2]	B	38.60[S2]	19.65	6.00	-3.34[S3]	-2.79[S4]	-0.335[S4]	
Slope: Soil → Blood[2]	F				8.06[S2]	7.54[1T]	9.13[S1]	6.28[S1]
INTERCEPT[3]	C_g	211[S3]	152[1T]	867[S3]	182[S2]	180[1T]	885[S2]	182[S3]
Slope: Soil → Dust[4]	D	0.175[1T]	0.267[S1]	0.894[1T]	0.240[S1]	0.195	0.833[1T]	0.223[S1]
DUST LEAD LOAD for AGE 42+ MONTHS[1,2]								
INTERCEPT[1]	G_g	6.53[S2]	12.55[S4]	11.56[S4]	7.63[S4]	10.83[M]	7.69[S3]	6.89[S4]
Slope: Dust → Blood[6]	B	38.47	-0.51[S4]	-0.0016[S4]	-0.347[S4]	-0.462[M]	-0.00167[S4]	
Slope: Soil → Blood[2]	F				8.63[M]	2.15	9.04[S1]	6.58[S1]
INTERCEPT[3]	C_g	17.2	129.7	1703	16.2	111.6	1640	18.9
Slope: Soil → Dust[7]	D	0.087[1T]	0.068	-0.394	0.128[1T]	0.099	0.0073	0.116[1T]

[1]Units are μg/dL Pb in blood.
[2]Units are μg/dL Pb in blood per 1000 μg/g Pb in soil.
[3]Units are μg/g Pb in dust.
[4]Units are μg/g Pb in dust per μg/g in soil.
[5]Units are μg/g Pb in dust per μg/g in dust.
[6]Units are μg/dL Pb in blood per 1000 μg/m² in dust Pb load.
[7]Units are μg/m² Pb in dust per μg/g Pb in soil.
[8]Floor dust lead predicted from soil lead, only soil lead used to predict blood lead.

(-0.02 μg/dL per 1000 μg/g) and the soil lead coefficient was large (1.72 μg/dL), but not statistically significant. When both entry dust lead and soil lead concentration were used as predictors, the blood lead versus entry dust lead coefficient was large (26.4 μg/dL per 1000 μg/g) and extremely significant (P = 0.0001), but the blood lead versus soil lead concentration coefficient was also large and *negative* (-4.08 μg/dL per 1000 μg/g) and nearly significant, suggesting a serious collinearity problem in which neither coefficient was reliable. The relationship between floor dust lead concentration and soil concentration was large, 0.33, and statistically highly significant, as was the regression coefficient between entry dust lead concentration and soil lead, 0.31. The floor and entry dust lead loading coefficients were only somewhat less significant.

Table 5-3 shows somewhat similar results when the data were restricted to a subset with ages 42+ months. The best-fitting models for blood lead used entry dust lead concentration or mat dust lead loading. The blood lead versus entry dust lead concentration regression coefficient was large (19.65 μg/dL per 1000 μg/g lead in entry dust) but was not statistically significant, while the entry dust lead versus soil lead concentration coefficient of 0.267 was large and statistically significant. Floor dust lead concentration produced a somewhat worse fit to the blood lead data as measured by RMSE, but a statistically very significant regression coefficient between blood lead and floor dust lead concentration, 38.6 μg/dL per 1000 μg/g lead in floor dust.

The Round 1 blood lead and dust lead models evaluated here do not identify an across-the-board best model using any dust lead index, although floor dust lead appears adequate in most cases. Models using flood dust lead concentration and floor dust lead loading are compared in Table 5-4. The best fitting models are shown schematically in Figure 5-47.

The best fitting model of all tested, in the right column of Table 5-3, uses only soil lead to predict blood lead for children aged 42 months and older. The regression coefficients for blood lead versus soil lead are large and statistically significant, whether in simultaneous fitting of blood lead and floor dust lead concentration (6.28 μg/dL per 1000 μg/g lead in soil) or simultaneously fitting blood lead and floor dust lead loading (6.58 μg/dL per 1000 μg/g lead in soil). In the longitudinal models described below, we therefore used both floor dust lead concentration and soil lead concentration as predictors of blood lead lead for Round 1.

TABLE 5-4. PREABATEMENT CROSS-SECTIONAL STRUCTURAL EQUATION MODELS FOR CINCINNATI STUDY: FLOOR DUST

SEM EQUATION COEFFICIENTS		FLOOR DUST LEAD CONCENTRATION[1,2]							
		Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
INTERCEPT[1] G_{g1}		8.12[S4]	7.64[S4]	7.55[S4]	8.02[S4]	7.58[S3]	8.47[S3]	7.41[S4]	8.47[S3]
SLOPE	Floor → Blood[2]		4.17[S1]	4.10[S1]	0.70	4.43		4.65	-0.02
	Soil → Blood[2]	1.3		0.28	1.12		1.72		1.72
	Window → Blood[2]	0.190[S1]			0.16	-0.01			
INTERCEPT[3] G_{g2}		99.9[S2]	99.4[S2]	99.9[S2]	99.9[S2]	99.5[S3]	160.3[S3]	160.4[S4]	160.3[S4]
SLOPE	Soil → Floor[4]	0.2264[S3]	0.2272[S3]	0.2247[S3]	0.2260[S3]	0.2266[S3]	0.3308[S4]	0.3303[S4]	0.3308[S4]
	Window → Floor[5]	0.0458[S3]	0.0457[S3]	0.0458[S3]	0.0458[S3]	0.0457[S3]			

		FLOOR DUST LEAD LOADING				
		Model 9	Model 10	Model 11	Model 12	Model 13
INTERCEPT[1] G_{g1}		8.40[S4]	8.29[S4]	8.54[S4]	8.80[S4]	8.65[S4]
SLOPE	Floor → Blood[6]		-1.60		0.93	-0.25
	Soil → Blood[2]	1.26	2.56	1.58		2.18
	Window → Blood[6]	0.003	0.0117			
INTERCEPT[3] G_{g2}		-3.83	-3.98	15.05	15.54	15.02
SLOPE	Soil → Dust[7]	0.0898[S2]	0.0904[S3]	0.1486[S2]	0.1464[S2]	0.1491[S2]
	Window → Floor[8]	0.00771[S4]	0.00772[S4]			

[1]Units are μg/dL Pb in blood.
[2]Units are μg/dL Pb in blood per 1000 μg/g Pb in soil.
[3]Units are μg/g Pb in dust.
[4]Units are μg/g Pb in dust per μg/g in soil.
[5]Units are μg/g Pb in dust per μg/g in dust.
[6]Units are μg/dL Pb in blood per 1000 μg/m^2 in dust Pb load.
[7]Units are μg/m^2 Pb in dust per μg/g Pb in soil.
[8]Units are μg/m^2 Pb in dust per μg/m^2 Pb in dust.

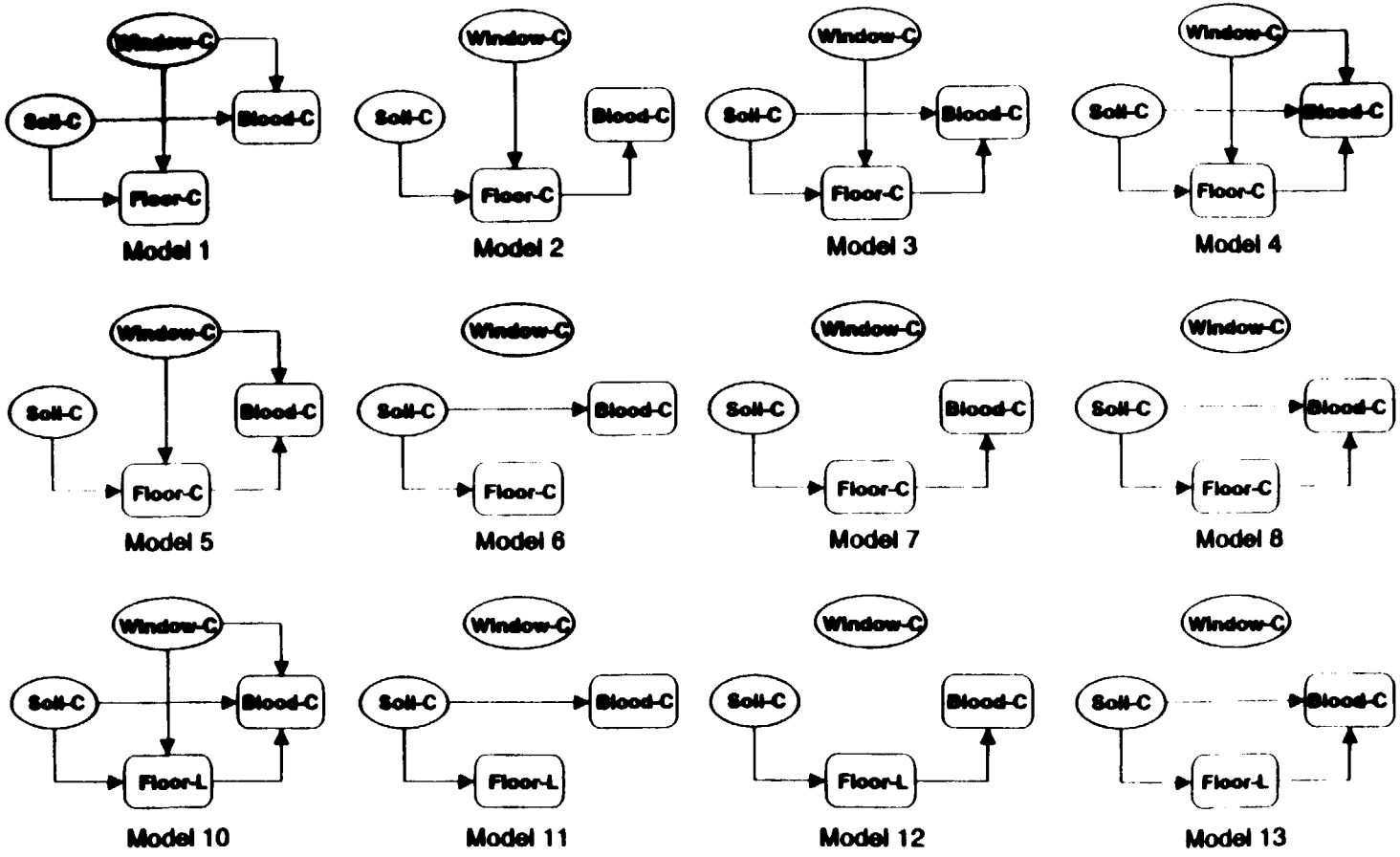

Figure 5-47. Pathway diagram of twelve different cross-sectional structural equation models for Round 1 of the Cincinnati study. Arrows show regression models for blood lead and floor dust lead concentrations. Oval figures show soil lead concentration and window dust lead concentration as independent variables. Regression coefficients are shown in Table 5-3.

The Cincinnati cross-sectional structural equation models are shown in Table 5-4. The floor dust lead model is extended by including window lead as a predictor of both floor dust lead and blood lead, analogous to the Boston models. While the various models shown in Table 5-4 provide only a slight additional improvement in Round 1 blood lead, they provide a significant improvement in the prediction of Round 1 floor dust lead concentration. In model 4 in which floor dust lead concentration, window dust lead concentration, and soil lead concentration are all used as predictors, none of these variables is statistically significant. In combinations using one or two of the variables, models 1, 2, 3, and 5 were best. Model 1 shows a statistically significant relationship between window dust and blood lead, whereas the soil lead coefficient was not statistically significant. In Models 2 and 3, there were statistically significant relationships between blood lead and floor dust lead concentration, 4.17 μg/dL per 1000 μg/g lead in floor dust in Model 2. The Model 2 regression coefficients between floor dust lead concentration and soil lead concentration, 0.227, and between floor dust lead concentration and window dust lead concentration, 0.0457, were highly significant. This was therefore the starting point for the longitudinal models described in Section 5.6. Additional analyses were done using floor dust lead loading, but the regression coefficients for floor dust lead concentration were generally more stable and more significant. Note also that Models 7 and 8 are similar to Models 2 and 3 respectively, except that window dust lead is not used as a predictor of floor dust lead. The floor dust lead regression coefficient in Model similar in magnitude to that in Model 2, but is not statistically significant. The floor dust lead and soil dust lead regression coefficients in Model 8 are quite different from those in Model 3, and are not statistically significant, whereas the blood lead vs floor dust lead concentration regression coefficient in Model 3 is statistically significant. This table demonstrates the importance of simultaneous fitting of pathway model components. Models 9-13 used floor dust lead loading, which in general was not as good a predictor of blood lead.

5.5 COMPARISON BY REPEATED MEASURES ANALYSIS

Several approaches are evaluated for analyzing the longitudinal data from the three cities using "repeated measures" models. In many cases, the ability to identify differences

among interventions was greatly improved by including covariates in the analyses. For example, child blood lead is known to change with age. When age is included as a covariate, some of the variation in blood lead differences before and after abatement can be attributed to the age of the child when the abatement was carried out. Controlling for the influence of age increases the ability to more accurately estimate the relationship between blood lead and other variables, such as soil lead. Similarly, the effect of abatement may depend on changes in proximate exposure variables such as house dust lead. The effects of changes in house dust lead may be different at different ages, however, so that other covariates that may be useful in the analyses include interactions between age, house dust lead, and treatment group.

The use of baseline preabatement environmental or demographic measurements for individual subjects as covariates allows one to proceed as if all groups had the same starting values. The use of differences in environmental measurements before and after abatement allows one to proceed as if individuals responded similarly to similar changes in lead exposure, which is a fundamental assumption in a remediation and intervention program. In general, differences in environmental indices before and after abatement were found to be no more predictive of blood lead changes than the absolute baseline or final values.

Repeated measures analyses can be carried out using standard statistical programs for analyses of general linear models. The PROC MIXED program in the SAS statistical package (SAS, 1992) was used for most of the analyses. Analyses of repeated measures models with time-varying covariates can be conveniently carried out using these programs. Repeated measures models with more than two phases or time points may require specific assumptions about time correlation structure in some programs, but no such assumptions are needed here when comparing outcomes at only two time points, pre- and postabatement.

5.5.1 Boston Repeated Measures Analysis of Variance (ANOVA)

The Boston repeated measures ANOVA results for all 150 children (excluding the two who became lead-poisoned) are shown in Tables 5-5 and 5-6. The repeated measures ANOVA coefficients of Table 5-5 and following are taken from equation 5-16 of Section 5.1.2, also repeated here for convenience.

TABLE 5-5. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BOSTON STUDY: EFFECT OF AGE ON REDUCTION IN BLOOD LEAD (E_r) BETWEEN ROUNDS 1 AND 3

Study Group		Age Group[1]			
		All Ages (N=150)	0-17 Months (N=19)	18-41 Months (N=100)	42+ Months (N=31)
Abate	Control	Response[2] (E_r)			
BOS SPI	BOS P-S	1.87[S2]	0.69	2.51[S2]	1.09
BOS PI-S	BOS P-S	0.33	-2.09	1.17[M]	-0.75
BOS SPI	BOS PI-S	1.54[S1]	2.78	1.33[1T]	1.84
		Log Response[3] (E_r)			
BOS SPI	BOS P-S	0.164[S2]	0.056	0.201[S2]	0.146
BOS PI-S	BOS P-S	0.028	-0.177	0.077	0.007
BOS SPI	BOS PI-S	0.136[S1]	0.233	0.124[1T]	0.140

[1]Age is age in months at time of Round 1 blood sample.
[2]Units are μg/dL Pb in blood.
[3]Units are log μg/dL Pb in blood.

TABLE 5-6. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BOSTON STUDY: EFFECT OF AGE ON REDUCTION IN BLOOD LEAD (E_r) BETWEEN ROUNDS 3 AND 4

Study Group		Age Group[1]			
		All Ages (N=147)	0-17 Months (N=18)	18-41 Months (N=98)	42+ Months (N=31)
Abate	Control	Response[2] (E_r)			
BOS P-S	BOS SPI	1.77[M]	1.12	0.09	3.71[M]
BOS PI-S	BOS SPI	3.80[S3]	4.33	3.39[S1]	3.76[M]
BOS PI-S	BOS P-S	2.03[1T]	3.21	2.49[1T]	0.05
		Log Response[3] (E_r)			
BOS P-S	BOS SPI	0.125	0.228	0.042	0.359
BOS PI-S	BOS SPI	0.310[S1]	0.401	0.299[1T]	0.278
BOS PI-S	BOS P-S	0.185[M]	0.173	0.257[M]	0.081

[1]Age is age in months at time of Round 1 blood sample.
[2]Units are μg/dL Pb in blood.
[3]Units are log μg/dL Pb in blood.

$$Y_{ir} = G_{gr} + H_{h(g)} + I_{i(gh)} + e_{ir} \quad (5\text{-}16)$$

Effect sizes can be calculated very similarly with the methods used in this report. The simplest effect size calculation, comparing Group g to Group h across rounds 1 and r, is the difference

$$E_r = (G_{g1} - G_{gr}) - (G_{h1} - G_{hr}) \quad (5\text{-}20)$$

This is used in repeated measures ANOVA, and to calculate the intercept effect in RM ANCOVA.

All tables show the estimated effect of soil abatement or dust abatement groups versus reference groups. Table 5-5 shows the mean reduction in blood lead during Phase 1 of the Boston study, Round 1 to Round 3. The soil abatement group BOS SPI shows a highly significant (P = 0.0042) decrease in blood lead of 1.87 μg/dL relative to the control group BOS P-S, and a statistically significant decrease (P = 0.016) in blood lead of 1.54 μg/dL in the soil abatement group BOS SPI compared to the group BOS PI-S that received only dust abatement. The groups BOS PI-S and BOS P-S did not show significantly different (P = 0.62) changes in blood lead between Rounds 1 and 3. Table 5-5 also shows similarly significant (P = 0.0064) reductions in the logarithm of blood lead, with the effect size of soil abatement relative to control of 0.164 and relative to dust abatement of 0.136 (P = 0.021). This corresponds to estimated effects on a percentage basis of 17.8% for BOS SPI versus BOS P-S and 14.6% for BOS SPI versus BOS PI-S. This percentage is calculated from the log transformed data as a function of the geometric mean (GM), where

$$GM_{g1} = e^{m_1}$$

$$GM_{g2} = e^{m_2}$$

$$PERCENT\ CHANGE = \left(\frac{GM_{g2}}{GM_{g1}} - 1\right)100 \qquad (5\text{-}21)$$

$$= \left(e^{m_2 - m_1} - 1\right)100$$

Table 5-6 shows the mean reduction in blood lead during Phase 2 of the Boston study, Round 3 to Round 4. Two groups received soil and dust abatement during Phase 2, BOS PI-S and BOS P-S. Group BOS SPI received no further abatement and was an appropriate reference group for Phase 2 comparison. The soil abatement group BOS PI-S shows a highly significant (P = 0.0006) decrease in blood lead of 3.80 μg/dL relative to the reference group BOS SPI, and a marginally significant (P = 0.12 two-tailed, 0.061 one-tailed) decrease in blood lead of 1.77 μg/dL in the soil abatement group BOS P-S compared to the reference group BOS SPI that received no further abatement in Phase 2. The groups BOS PI-S and BOS P-S showed near-significantly (P = 0.079) different changes in blood lead between Rounds 3 and 4, with group BOS PI-S that had received dust abatement in Phase 1 showing an additional 2.03 μg/dL reduction compared to group BOS P-S that did not receive dust abatement. Table 5-6 also shows similarly significant (P = 0.022) reductions in the logarithm of blood lead, with the effect size of soil abatement in BOS PI-S relative to BOS SPI of 0.310 and BOS P-S relative to BOS SPI of 0.125 (NS, P = 0.37). There was also an indication of a difference between BOS P-S and BOS PI-S of 0.185, but it was very marginally significant (P = 0.18) although in the expected direction. This corresponds, on a percentage basis, to an estimated Phase 2 effect of BOS PI-S relative to BOS SPI of 36.3%.

Age Effects in Boston

Tables 5-5 and 5-6 also show a breakdown of the Boston ANOVA results by age category. Because of greatly reduced sample sizes, none of the effect size estimates in the youngest age group (9 to 17 months at Round 1) are statistically significant, even though they

are large in magnitude and somewhat similar to those in older children. The largest subset of Boston children, ages 18-41 months at Round 1, showed an even larger effect of soil abatement than did the whole group. Table 5-5 shows a mean effect of BOS SPI versus BOS P-S of 2.51 μg/dL (P = 0.0020). Table 5-5 also shows some differentiation among treatments, with a nearly significant (P = 0.077 two-tailed, 0.038 one-tailed) effect of 1.33 μg/dL between the Phase 1 soil abatement group BOS SPI and dust abatement group BOS PI-S. The log-transformed results for Phase 1 in Table 5-5 are also highly significant (P = 0.0084) in the 18-41 month age group and somewhat larger than for the whole group, with an effect size of 0.201 or 22.3% reduction in blood lead in the BOS SPI group compared to the BOS P-S control group. Effect sizes in the age group 42+ months are smaller and not statistically significant, which may also be due to a small sample size.

The Phase 2 results are shown in Table 5-6. The effects are all in the expected direction. The group BOS PI-S that had abatements in both Phase 1 and Phase 2 showed the greatest reductions in blood lead. The group BOS P-S that received Phase 2 soil abatement showed a greater Phase 2 reduction in blood lead lead than the group BOS SPI that received Phase 1 soil abatement, but not further remediation in Phase 2. All of the effect size estimates are positive, at all ages. In the youngest age group, there are no statistically significant effects, but the magnitude of the blood lead reductions is large and similar to those in older children. In the 18 to 41 month age group, the significant effects are slightly smaller than for the group as a whole. The Phase 2 soil abatement group BOS PI-S had a significantly larger (P = 0.014) decrease in blood lead of 3.39 μg/dL than did BOS SPI, and a marginally greater reduction of 2.49 μg/dL (P = 0.076) than did group BOS P-S. The log-transformed analysis shown in Table 5-6 found somewhat less significant (P = 0.074) effects for 18-41 month old children, with an effect of 0.299 for BOS PI-S versus BOS SPI, and an effect of 0.257 (P = 0.13) for BOS PI-S versus BOS P-S. There was also some indication in Table 5-6 of an effect of soil lead abatement at Phase 2 in the older children, ages 42+ months, amounting to 3.71 μg/dL (P = 0.12) for BOS P-S versus BOS SPI, and 3.76 μg/dL (P = 0.10) for BOS PI-S versus BOS SPI. The results for older children were large but not significant (P = 0.21) on a log scale, amounting to 0.359 for BOS P-S versus BOS SPI, and 0.278 (P = 0.31) for BOS PI-S versus BOS SPI in Table 5-6.

Race/Ethnicity Effects in Boston

The analysis of subgroups of children clearly identified as Afro-American and as non-Black did not find large differences in overall response in Phase 1, although other analyses using analysis of covariance (ANCOVA) methods suggest some ethnic differences in response to environmental lead exposure. Table 5-7 shows that there was a significant (P = 0.050 two-tailed, 0.025 one-tailed) effect of 1.91 μg/dL comparing BOS SPI to BOS P-S from Rounds 1 to 3. The logarithmic analysis in Table 5-7 found a nearly significant (P = 0.064) effect of 0.163 or 17.7% reduction from soil abatement. The effects for non-blacks were smaller and only marginally significant, although not significantly different in magnitude.

Results in Phase 2 of the study were substantially larger. Both of the Phase 2 soil abatement groups in Table 5-7 showed substantial and statistically significant reductions relative to the Phase 2 non-abatement group BOS SPI. The effect for BOS P-S versus BOS SPI was 3.65 μg/dL (P = 0.014), much larger than the overall group effect, and the effect for BOS PI-S versus BOS SPI was 4.46 μg/dL (P = 0.0044). However, the log-transformed analyses in Table 5-7 showed less significant (P = 0.070) changes, with only the BOS PI-S versus BOS SPI effect of 0.400 nearly significant. None of the effects for non-Black children were statistically significant, although this may be a consequence of the relatively small number of children identified in this subgroup, N = 32, since the estimated effect of BOS PI-S versus BOS P-S of 4.04 μg/dL was relatively large.

Gender Effects in Boston

Table 5-7 shows large effects in male children. The Phase 1 effect of both soil abatement and dust abatement appear to be very similar and significant, 2.19 μg/dL for BOS SPI versus BOS P-S and 2.08 μg/dL (P = 0.016) for BOS PI-S versus BOS P-S (P = 0.020). The logarithmic analyses in Table 5-7 find even more significant (P = 0.0099) effects for males, 0.197 for BOS SPI versus BOS P-S and 0.194 for BOS PI-S versus BOS P-S. The Phase 1 effects for female children are smaller and only marginally significant, 1.45 μg/dL for BOS SPI versus BOS P-S (P = 0.13). The male-female differences are not significant, possibly due to larger uncertainty in the estimated female effect sizes.

TABLE 5-7. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BOSTON STUDY: EFFECT OF RACE OR SEX

Study Group			Group				
		Rounds 1-3:	All (N=150)	Black (N=75)	Nonblack (N=32)	Male (N=80)	Female (N=70)
		Rounds 3-4:	(N=147)	(N=74)	(N=32)	(N=78)	(N=69)
Abate	Control		Reduction in Blood Lead (E_T) Between Rounds 1 and 3[1]				
BOS SPI	BOS P-S		1.87^{S2}	1.91^{1T}	1.54	2.19^{S1}	1.45^{M}
BOS PI-S	BOS P-S		0.33	0.78	-0.18	0.11	0.52
BOS SPI	BOS PI-S		1.54^{S1}	1.13	1.72^{M}	2.08^{S1}	0.93
			Reduction in Blood Lead (E_T) Between Rounds 3 and 4[1]				
BOS P-S	BOS SPI		1.77^{M}	3.65^{S1}	-1.86	1.65	2.19
BOS PI-S	BOS SPI		3.80^{S3}	4.46^{S2}	2.18	3.02^{S1}	5.00^{**}
BOS P-S	BOS P-S		2.03^{1T}	0.81	4.04^{M}	1.37	2.80^{1T}
			Reduction in Log Blood Lead (E_T) Between Rounds 1 and 3[2]				
BOS SPI	BOS P-S		0.164^{S2}	0.163^{1T}	0.070	0.197^{S2}	0.130^{M}
BOS PI-S	BOS P-S		0.028	0.092	-0.105	0.002	0.050
BOS SPI	BOS PI-S		0.136^{S1}	0.070	0.175^{M}	0.194^{S2}	0.080
			Reduction in Log Blood Lead (E_T) Between Rounds 3 and 4[2]				
BOS P-S	BOS SPI		0.125	0.206	0.038	0.176	0.080
BOS PI-S	BOS SPI		0.310^{S1}	0.400^{1T}	0.144	0.173	0.470^{1T}
BOS P-S	BOS P-S		0.185^{M}	0.194	0.106	-0.003	0.390^{M}

[1]Units are μg/dL Pb in blood.
[2]Units are log μg/dL Pb in blood.

The Phase 2 analyses shown in Table 5-7 find larger and more significant effects in female children. The Phase 2 effect of soil abatement in BOS PI-S versus no abatement in BOS SPI is 3.00 μg/dL ($P = 0.05$) in males, but 5.00 μg/dL in females ($P = 0.0039$). There may also be a marginally significant difference ($P = 0.093$) between female children in the two Phase 2 soil abatement groups, with a somewhat greater effect of 2.80 μg/dL in BOS PI-S (that had Phase 1 dust abatement) compared to BOS P-S (no Phase 1 abatement). However, the statistical significance of the log-transformed data in Table 5-7 was much lower, with no significant Phase 2 abatement effects for males and less significant effects for females in BOS PI-S versus BOS SPI and in BOS PI-S versus BOS P-S compared with the

non-transformed data analyses shown in Table 5-7. The male-female differences are not significant, possibly due to larger uncertainty in the estimated female effect sizes.

Blood Lead Truncation Effects in Boston

The design of the Boston study involved some truncation of the range of starting values of blood lead concentration. One way of assessing the possible effect of this design choice is to compare the results of the analyses with the results of similar analyses of the Boston data set using truncated subsets of the data. After preliminary assessment to determine the number of observations with different truncation levels, the following truncated data sets were selected:

Full data set, 7 to 24 μg/dL;

Minimal data set, 10 to 19 μg/dL;

Upper truncation, 7 to 19 μg/dL;

Lower truncation, 10-24 μg/dL.

The minimal data set includes only children who would have been considered lead-burdened at the beginning of the study (blood lead at least 10 μg/dL) and excludes those children whose blood lead concentrations were at least 20 μg/dL and whose residences might have been considered as appropriate for environmental intervention on the basis of the blood lead finding. The upper truncation adds children with somewhat lower blood leads to the minimal data set, and the lower truncation adds children with higher blood leads to the minimal data set.

The truncated data sets for children ages 9 to 17 months at Round 1 did not show any significant effects, probably due to the very small sample sizes, and are not discussed further. The Phase 1 results for children of ages 18 to 41 months are shown in Table 5-8. The highly significant whole-sample effect of 2.51 μg/dL for BOS SPI versus BOS P-S in Phase 1 is largely insensitive to the truncation, increasing to 2.67 μg/dL (P = 0.013) in the minimal data set and 2.73 μg/dL (P = 0.0070) in the lower truncation data set, and decreasing only to 2.42 μg/dL (P = 0.0039) in the upper truncation data set. The near significant effect of BOS SPI relative to BOS PI-S is increased in all of the truncation data sets, with somewhat lower significance (P = 0.059) due to the smaller sample size except for the upper truncation, where the effect size is 1.53 μg/dL. The log-transformed analyses

TABLE 5-8. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BOSTON STUDY: EFFECT OF TRUNCATION ON REDUCTION IN BLOOD LEAD (E_T) BETWEEN ROUNDS 1 AND 3

Study Group		Truncation Category			
	Age 18-41:	7-24 μg/dL[1] (N = 100)	10-19 μg/dL (N = 67)	7-19 μg/dL (N = 92)	10-24 μg/dL (N = 75)
	Age 42-52:	(N = 31)	(N = 16)	(N = 29)	(N = 18)
Abate	Control	Change in Blood Lead for Age Group 18-41 Months[2]			
BOS SPI	BOS P-S	2.51[S2]	2.67[S1]	2.42[S2]	2.73[S2]
BOS PI-S	BOS P-S	1.17[M]	0.96	0.89	1.29[M]
BOS SPI	BOS PI-S	1.33[1T]	1.71[M]	1.53[1T]	1.43[M]
		Change in Blood Lead for Age Group 42-52 Months[2]			
BOS SPI	BOS P-S	1.09	0.50	1.45	0.00
BOS PI-S	BOS P-S	-0.75	-1.58	-0.20	-2.25
BOS SPI	BOS PI-S	1.84	2.08	1.65	2.25
		Change in Log Blood Lead for Age Group 18-41 Months[3]			
BOS SPI	BOS P-S	0.201[S2]	0.212	0.205[S1]	0.203[S1]
BOS PI-S	BOS P-S	0.077	0.059	0.068	0.068
BOS SPI	BOS PI-S	0.124[1T]	0.153[M]	0.137[1T]	0.135[M]
		Change in Log Blood Lead for Age Group 42-52 Months[3]			
BOS SPI	BOS P-S	0.146	0.042	0.176[M]	0.010
BOS PI	BOS P-S	0.007	-0.154	0.035	-0.180
BOS SPI	BOS PI-S	0.140	0.196	0.142	0.191

[1]In the Boston study, children with screening blood lead concentrations below 7 μg/dL and above 24 μg/dL were excluded from the study for reasons discussed in Chapter 4.
[2]Units are μg/dL Pb in blood.
[3]Units are log μg/dL Pb in blood.

show a similar pattern, with larger but less significant effects of BOS SPI versus BOS P-S and BOS SPI versus BOS PI-S in the truncated data sets.

The Phase 2 results for children of ages 18-41 months are shown in Table 5-9. The significant whole-sample effect of 3.39 μg/dL for BOS PI-S versus BOS SPI in Phase 2 is more sensitive to the truncation, increasing to 3.89 μg/dL (P = 0.033) in the minimal data set and to 4.85 μg/dL (P = 0.0040) in the lower truncation data set, and decreasing to 2.56 μg/dL (P = 0.079) in the upper truncation data set. However, the effect of the other Phase 2 soil abatement group BOS P-S versus BOS SPI, which was not at all significant in

TABLE 5-9. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BOSTON STUDY: EFFECT OF TRUNCATION ON REDUCTION IN BLOOD LEAD (E_T) BETWEEN ROUNDS 3 AND 4

Study Group		Truncation Category			
		7-24 μg/dL[2] (N = 31)	10-19 μg/dL (N = 16)	7-19 μg/dL (N = 29)	10-24 μg/dL (N = 18)
Abate	Control	Change in Blood Lead for Age Group 18-41 months[2]			
BOS P-S	BOS SPI	0.90	2.10	0.53	2.40[M]
BOS PI-S	BOS SPI	3.39[S1]	3.89[S1]	2.56[1T]	4.85[S2]
BOS PI-S	BOS P-S	2.49[1T]	1.79	2.03[M]	2.45[M]
		Change in Log Blood Lead for Age Group 18-41 Months[3]			
BOS P-S	BOS SPI	0.042	0.211	0.013	0.226[M]
BOS PI-S	BOS SPI	0.299[1T]	0.372[S1]	0.266[M]	0.414[S2]
BOS P-S	BOS P-S	0.257[M]	0.161	0.252[M]	0.188

[1]In the Boston study, children with screening blood lead concentrations below 7 μg/dL and above 24 μg/dL were excluded from the study for reasons discussed in Chapter 4.
[2]Units are μg/dL Pb in blood.
[3]Units are log μg/dL Pb in blood.

the whole sample, is larger and very marginally significant (P = 0.17) in the truncated sample with children 7 to 9 μg/dL omitted. The nearly significant effect of BOS PI-S relative to BOS P-S in Table 5-9 is decreased in all of the truncation data sets, with much lower significance due to the smaller sample size. The log-transformed analyses show a similar pattern. The nearly significant effect of BOS PI-S versus BOS SPI in the whole sample is larger and much more significant (P = 0.0093) in the lower truncated sample. The non-significant effect of BOS P-S versus BOS SPI in the whole sample is similarly larger and more significant (P = 0.17) in the lower truncated sample. The Phase 1 results for children of ages 42+ months are shown in Table 5-8. None of the effects is even marginally significant, and this is not substantially changed by truncating the data set. The log-transformed analyses show a similar pattern, with the only change in significance class occurring in BOS SPI versus BOS P-S with P = 0.25 for the whole data set and P = 0.19 in the upper truncation data set.

The Phase 2 results for children of ages 42+ months are shown in Table 5-9. The marginally significant whole-sample effect of 3.76 μg/dL for BOS PI-S versus BOS SPI in Phase 2 is somewhat sensitive to the truncation, increasing to 5.76 μg/dL (P = 0.13) in the

minimal data set and to 5.90 μg/dL (P = 0.067) in the lower truncation data set, and decreasing only to 3.70 μg/dL (P = 0.13) in the upper truncation data set. The marginally significant effect of BOS P-S relative to BOS SPI is almost unchanged in all of the truncation data sets, with lower significance due to the smaller sample size. The log-transformed analyses show a similar pattern, with no significant effects of BOS PI-S versus BOS SPI nor BOS P-S versus BOS SPI in the truncated data sets.

In summary, there appears to be some sensitivity to truncation of the data set. There were a number of situations in which larger and more significant results were found in the truncated data set, particularly when children with initial blood lead concentrations less than 10 μg/dL were omitted. The effects of truncation were noted in both Phase 1 and Phase 2 analyses.

5.5.2 Cincinnati Repeated Measures Analysis of Variance (ANOVA)

The analyses are based on 223 children whose blood lead measurements were taken in both Rounds 1 and 4. As noted above, the sample sizes reported are the maximum number of children who could have been used in the analyses. Due to missing values in covariates or classification variables, the actual number used is generally somewhat smaller. The effect size comparisons are based on neighborhood-by-neighborhood comparisons for the 5 neighborhoods with sufficient numbers of follow-up measurements to allow comparisons. The Phase 1 dust abatement neighborhoods are CIN I-SE(D) and CIN I-SE(F); CIN I-SE(B) was omitted. The no-treatment or control neighborhoods for both Phase 1 (rounds 1 to 4) and Phase 2 (rounds 4 to 7) were CIN NT(G) and CIN NT(M). The Phase 1 soil abatement neighborhood was CIN SEI(P), which also received no abatement in Phase 2 of the Cincinnati study. Comparisons are carried out among neighborhoods in different treatment groups, and sometimes between neighborhoods within each group.

Table 5-10 shows the effect size for blood lead between Round 1 and Round 4. One of the largest differences in the study is the difference between the two control or no-treatment neighborhoods, CIN NT(G) and CIN NT(M), with blood lead in CIN NT(G) decreasing 3.58 μg/dL more than in CIN NT(M) between Round 1 and Round 4 (P = 0.073). Blood lead in the soil abatement neighborhood, CIN SEI(P), decreased a little more than in the no-treatment neighborhood of CIN NT(M), 1.02 μg/dL (P = 0.60), but blood lead in the

TABLE 5-10. REPEATED MEASURES ANALYSIS OF VARIANCE FOR CINCINNATI STUDY: EFFECT OF AGE BETWEEN ROUNDS 1 AND 4

Study Group		Age Group[1]			
		All Ages (N=223)	9-17 Months (N=69)	18-41 Months (N=80)	42+ Months (N=70)
Abate	Control	Reduction in Blood Lead (E_τ) Between Rounds 1 and 4[2]			
CIN NT (G)	CIN NT (M)	3.58^{1T}	13.10^{S3}	9.21^{1T}	-2.57^{M}
CIN SEI (P)	CIN NT(G)	-2.56^{S1}	-5.62^{S1}	-0.97	-1.97^{M}
CIN SEI (P)	CIN NT (M)	1.02	7.48^{1T}	8.24^{M}	-4.55^{S1}
CIN SEI (P)	CIN I-SE (D)	-2.43^{S1}	-1.57	0.10	-4.34^{S2}
CIN SEI (P)	CIN I-SE (F)	-1.20	-0.91	-0.26	-2.99^{1T}
CIN I-SE (D)	CIN NT (G)	-0.14	-4.05^{M}	-1.07	2.36^{M}
CIN I-SE (D)	CIN NT (M)	3.44^{1T}	9.05^{S1}	8.14^{M}	-0.21
CIN I-SE (F)	CIN NT (G)	-1.37	-4.70^{1T}	-0.70	1.02
CIN I-SE (F)	CIN NT (M)	2.21	8.40^{S1}	8.51^{1T}	-1.55
		Reduction in Log Blood Lead (E_τ) Between Rounds 1 and 4[3]			
CIN NT (G)	CIN NT (M)	0.615^{S2}	1.903^{S4}	0.804	-0.129
CIN SEI (P)	CIN NT(G)	-0.403^{S3}	-0.775^{S2}	-0.199	-0.346^{S1}
CIN SEI (P)	CIN NT (M)	0.212	1.128^{S2}	0.605^{M}	-0.475^{S1}
CIN SEI (P)	CIN I-SE (D)	-0.241^{1T}	-0.142	0.007	-0.409^{S2}
CIN SEI (P)	CIN I-SE (F)	-0.170	-0.187	-0.048	-0.336^{1T}
CIN I-SE (D)	CIN NT (G)	-0.163	-0.634^{S1}	-0.206	0.063
CIN I-SE (D)	CIN NT (M)	0.453^{S1}	1.269^{S3}	0.598^{M}	-0.065
CIN I-SE (F)	CIN NT (G)	-0.233^{M}	-0.589^{S1}	-0.152	-0.010
CIN I-SE (F)	CIN NT (M)	0.383^{1T}	1.314^{S3}	0.652^{M}	-0.138

[1]Age is age in months at time of Round 1 blood sample.
[2]Units are μg/dL Pb in blood.
[3]Units are log μg/dL Pb in blood.

dust abatement neighborhoods decreased much more, 3.44 μg/dL for CIN I-SE(D) versus CIN NT(M) (P = 0.08) and 2.21 μg/dL for CIN I-SE(F) versus CIN NT(M) (P = 0.30). On the other hand, blood lead decreased much less in CIN SEI(P) than in the other

no-treatment neighborhood, CIN NT(G), -2.56 μg/dL (P= 0.036). In fact, CIN SEI(P) decreased much less than either of the dust abatement neighborhoods of CIN I-SE(D) (-2.43 μg/dL, P = 0.049) or CIN I-SE(F) (-1.20 μg/dL, P = 0.36). A negative sign on effect size means that the decrease was smaller in the comparison neighborhood than in the reference neighborhood, which is generally contrary to what was expected in one-tailed tests.

The log-transformed blood lead analyses for Phase 1 shown in Table 5-10 exhibit the same pattern, but with much more significant effects. The largest effect in Table 5-10 is the difference between the two no-treatment neighborhoods, 0.615 on a log scale (P = 0.0032). This corresponds to a percentage difference of 85% in blood lead reduction between Round 1 and Round 4. The soil abatement neighborhood of CIN SEI(P) showed a significantly smaller reduction compared to CIN NT(G), -0.403 (P = 0.0016). While CIN SEI(P) showed a reduction of 0.212 or 23.6% compared to CIN NT(M), the effect was not significant (P = 0.30). Both CIN I-SE(D) and CIN I-SE(F) showed significant or near-significant improvements over CIN NT(M), respectively 0.453 (P = 0.03) and 0.383 (P = 0.07), but decreased less than the other control neighborhood, CIN NT(G).

The results for Phase 2 are shown in Table 5-11. The only effect of even marginal significance is the difference between the two control neighborhoods. However, the effect is in the opposite direction to the Phase 1 difference between CIN NT(G) and CIN NT(M), - 2.55 μg/dL. The log-transformed analyses for Phase 2 shown in Table 5-11 show a similar pattern, but with greater significance. The difference between CIN NT(G) and CIN NT(M) was larger in Phase 2, -0.334 on a log scale (P = 0.062), or 40%. While neither CIN SEI(P) nor CIN NT(G) received abatement during Phase 2, blood lead in CIN SEI(P) decreased more than in CIN NT(G) by 0.168 or 18.3% (P = 0.126), and less than in CIN NT(M) by -0.166 (P = 0.34).

Age Effects

Tables 5-10 through 5-11 also show age-stratified analyses. Unlike Boston and Baltimore, the Cincinnati study had almost the same number of children in each of the Round 1 age categories 9 to 17 months, 18 to 41 months, and 42+ months. In the youngest age group, the differences identified earlier were much larger and much more significant. The difference between CIN NT(G) and CIN NT(M) during Phase 1 was 13.1 μg/dL

TABLE 5-11. REPEATED MEASURES ANALYSIS OF VARIANCE FOR CINCINNATI STUDY: EFFECT OF AGE BETWEEN ROUNDS 4 AND 7

Study Group		Age Group[1]			
		All Ages (N = 223)	9-17 Months (N = 69)	18-41 Months (N = 80)	42+ Months (N = 70)
Abate	Control	Reduction in Blood Lead (E_r) Between Rounds 4 and 7[2]			
CIN NT (G)	CIN NT (M)	-2.55[M]	-4.56	-1.75	NC
CIN SEI (P)	CIN NT(G)	0.85	1.06	0.27	
CIN SEI (P)	CIN NT (M)	-1.70	-3.50	-1.48	
CIN SEI (P)	CIN I-SE (D)	-0.01	-1.13	0.78	
CIN SEI (P)	CIN I-SE (F)	0.72	1.42	0.23	
		Reduction in Log Blood Lead (E_r) Between Rounds 4 and 7[3]			
CIN NT (G)	CIN NT (M)	-0.334[1T]	NC	-0.188	-0.304[M]
CIN SEI (P)	CIN NT(G)	0.168[M]		0.128	0.210[M]
CIN SEI (P)	CIN NT (M)	-0.166		-0.059	-0.094
CIN SEI (P)	CIN I-SE (D)	0.033		0.075	0.123
CIN SEI (P)	CIN I-SE (F)	0.047		0.062	0.103

[1]Age is age in months at time of Round 1 blood sample.
[2]Units are μg/dL Pb in blood.
[3]Units are log μg/dL Pb in blood.

(P = 0.0014) as seen in Table 5-10. CIN SEI(P) was significantly less reduced than CIN NT(G), -5.6 μg/dL (P = 0.020) in the youngest age group, and reduced significantly more than in CIN NT(M), 7.5 μg/dL (P = 0.053). All of the other effect sizes compared to CIN NT(G) and CIN NT(M) were larger, in the same direction as the whole-group analyses, and much more significant in this age group. Findings were much more significant for the log-transformed analyses shown in Table 5-10. The two control groups showed the largest relative differences, with P < 0.0001. The estimated Phase 1 effect of CIN SEI(P) versus CIN NT(M) was 1.128 or 200% (P = 0.0098), and CIN SEI(P) versus CIN NT(G) was -0.775 (P = 0.0046). CIN I-SE(D) and CIN I-SE(F) also had significantly more reduction than CIN NT(M), and significantly less than CIN NT(G) during Phase 1.

Phase 1 effects for 18-41 month old children were consistently much smaller and much less significant than for 9-17 month old children. The most nearly significant Phase 1 results for 18-41 month old children were differences between CIN NT(G) and CIN NT(M) of 9.21 μg/dL (P = 0.072) and between CIN I-SE(F) and CIN NT(M) of 8.41 μg/dL

(P = 0.095). The CIN NT(G)-CIN NT(M) effect for Phase 1 was also nearly significant for 18-41 month children on a log scale, shown in Table 5-10 (P = 0.067).

Phase 1 effects for children of ages at least 42 months showed a very different pattern than for the younger children. Blood lead decreased less in CIN SEI(P) than in any other neighborhood. The *negative* effect of CIN SEI(P) versus CIN NT(G) was -1.97 μg/dL (P = 0.20), of CIN SEI(P) versus CIN NT(M) was -4.55 μg/dL (P = 0.017), of CIN SEI(P) versus CIN I-SE(D) was -4.34 μg/dL (P = 0.0028), and of CIN SEI(P) versus CIN I-SE(F) was -2.99 μg/dL (P = 0.082). Even the sign of the CIN NT(G)-CIN NT(M) difference was negative in the oldest age group. Table 5-10 shows the same pattern of Phase 1 effects using log blood lead of the older children, with similar statistical significance.

Phase 2 results were consistently non-significant within each age stratum, as shown in Table 5-11.

Effects of Blood Lead Truncation in Cincinnati

The range of blood lead concentrations in the Cincinnati study was not constrained by study design and was much larger than in the Boston study. Therefore, truncation of Cincinnati Round 1 blood lead to the corresponding Boston range (7 to 24 μg/dL) greatly reduced the sample size of the truncated data set. For children age 9 to 17 months, the sample size was reduced from 69 children for the whole data set to 33 in the truncated data set, with only 15 children remaining in the data set truncated to 10 to 19 μg/dL. Large and highly significant differences between CIN NT(G) and CIN NT(M), CIN NT(G), and CIN SEI, CIN NT(M) and CIN SEI that were found in the whole sample of 9 to 17 month old children completely lost statistical significance in the truncated samples, and their results are not reported here.

Fewer children were lost to truncation for ages 18-41 months, and the findings were statistically significant. Table 5-12 shows the results for Phase 1 effects. The effect size for CIN NT(G) versus CIN NT(M) Phase 1 blood lead reduction in the full sample was 9.21 μg/dL (P = 0.072), which was the same size but less significant (P = 0.10) in the 7 to 24 μg/dL truncation. However, the effect size for CIN NT(G) versus CIN NT(M) was larger and more significant in the smaller data sets: 11.76 μg/dL (P = 0.053) in the 10 to

TABLE 5-12. REPEATED MEASURES ANALYSIS OF VARIANCE FOR CINCINNATI STUDY: EFFECT OF TRUNCATION BETWEEN ROUNDS 1 AND 4

Study Group Abate Versus Control		Truncation Category			
		All	7-24 μg/dL	10-19 μg/dL	10-24 μg/dL
Abate	Control	Reduction in Blood Lead (E_T) Age 9-17 Months			
		(N=69)	(N=33)	(N=15)	
CIN NT (G)	CIN NT (M)	13.10[S3]	NC	NC	
CIN SEI (P)	CIN NT(G)	-5.62[S1]	-2.82	0.73	
CIN SEI (P)	CIN NT (M)	7.48[1T]	NC	NC	
CIN SEI (P)	CIN I-SE (D)	-1.57	-2.45	-0.86	
CIN SEI (P)	CIN I-SE (F)	-0.91	3.02	-2.83	
		Reduction in Blood Lead (E_T) Age 18-41 Months[1,2]			
		(N=80)	(N=67)	(N=38)	(N=43)
CIN NT (G)	CIN NT (M)	9.21[1T]	9.21[M]	11.76[1T]	11.11[1T]
CIN SEI (P)	CIN NT(G)	-0.97	-0.95	-3.52	-2.35
CIN SEI (P)	CIN NT (M)	8.24[M]	8.26[M]	8.24[M]	8.76[M]
CIN SEI (P)	CIN I-SE (D)	0.10	1.21	1.86	2.40
CIN SEI (P)	CIN I-SE (F)	-0.26	-0.81	-0.82	-0.33
		Reduction in Blood Lead (E_T) Age 42+ Months[1,2]			
		(N=70)	(N=47)	(N=31)	(N=36)
CIN NT (G)	CIN NT (M)	-2.57[M]	-1.61	-1.44	-1.49
CIN SEI (P)	CIN NT(G)	-1.97[M]	-2.73	-1.74	-1.70
CIN SEI (P)	CIN NT (M)	-4.55[S1]	-4.34[1T]	-3.17	-3.19
CIN SEI (P)	CIN I-SE (D)	-4.34[S2]	-5.17[S1]	-3.12	-4.64[M]
CIN SEI (P)	CIN I-SE (F)	-2.99[1T]	-2.06	-0.02	-0.07

[1]Units are μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.

19 μg/dL truncation sample, 11.11 μg/dL (P = 0.052) in the 10 to 24 μg/dL truncation sample. The CIN SEI versus CIN NT(G) effect size was larger and more negative in the truncated data sets, but the effects were still not statistically significant. The CIN SEI versus CIN NT(M) Phase 1 effect was little changed in either magnitude or statistical significance by truncation. Similar effects were obtained in the log-transformed analyses, not shown.

The Phase 2 results in Table 5-13 showed a few differences, but none of the truncation effects were statistically significant for 18-41 month old children.

TABLE 5-13. REPEATED MEASURES ANALYSIS OF VARIANCE FOR CINCINNATI STUDY: EFFECT OF TRUNCATION BETWEEN ROUNDS 1 AND 4

Study Group Abate Versus Control		Truncation Category		
		All	7-24 μg/L	10-19 μg/L
Abate	Control	Reduction in Log Blood Lead (E_T) Age 9-17 Months[1,2]		
		(N=69)	(N=33)	(N=15)
CIN NT (G)	CIN NT (M)	1.903[S4]	NC	NC
CIN SEI (P)	CIN NT(G)	-0.775[S2]	-0.153	-0.006
CIN SEI (P)	CIN NT (M)	1.128[S2]	NC	NC
CIN SEI (P)	CIN I-SE (D)	-0.142	-0.028	-0.126
CIN SEI (P)	CIN I-SE (F)	-0.187	0.421	-0.194
		Reduction in Log Blood Lead (E_T) Age 18-41 Months[1,2]		
		(N=80)	(N=67)	(N=38)
CIN NT (G)	CIN NT (M)	0.804[1T]	0.791[M]	NC
CIN SEI (P)	CIN NT(G)	-0.199[M]	-0.160	NC
CIN SEI (P)	CIN NT (M)	0.605[M]	0.631	NC
CIN SEI (P)	CIN I-SE (D)	0.007	0.075	NC
CIN SEI (P)	CIN I-SE (F)	-0.048	-0.091	NC
		Reduction in Log Blood Lead (E_T) Age 42+ Months[1,2]		
		(N=70)	(N=47)	(N=31)
CIN NT (G)	CIN NT (M)	-0.129	-0.064	NC
CIN SEI (P)	CIN NT(G)	-0.346[S1]	-0.332[M]	NC
CIN SEI (P)	CIN NT (M)	-0.475[S1]	-0.396[1T]	NC
CIN SEI (P)	CIN I-SE (D)	-0.409[S2]	-0.506[S1]	NC
CIN SEI (P)	CIN I-SE (F)	-0.336[1T]	-0.202	NC

[1]Units are log μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.

The effects of blood lead truncation in children of ages 42+ months, shown in Tables 5-14 and 5-15 was usually a modest reduction in the estimated size of the effect, but a large attenuation of statistical significance. This may be largely attributable to reduction in sample size. The Phase 1 reduction in blood lead for CIN SEI remained smaller than for any other neighborhood, but the statistical significance of the difference virtually disappeared. The difference between the two control neighborhoods also lost any statistical significance. The pattern of findings was also the same for log-transformed blood lead, not shown here. The Phase 2 results, which showed only a few small and marginally significant differences among neighborhoods in the whole sample, exhibited no significant differences in the truncated samples.

5.5.3 Baltimore Repeated Measures Analysis of Variance (ANOVA)

The Baltimore study carried out soil lead abatement (without interior dust lead abatement) in one neighborhood, Lower Park Heights. A small number of houses in this treatment group were not abated, almost all of which had no single soil sample above about 500 μg/g. In other words, the non-abated residences in Area 1 had a maximum soil lead concentration less than about 500 μg/g, whereas almost all of the abated residences had maximum soil lead above about 500 μg/g. For this reason, we used the control neighborhood of Walbrook Junction, where all properties had at least one soil measurement above 500 μg/g, and the unabated properties of lower Park Heights as separate controls rather than combining them as in the Baltimore report. The study group in Lower Park Heights is denoted BAL SP and the control groups are denoted BAL P1 for the Walbrook Junction group and BAL P2 for the low soil lead houses in Lower Park Heights. As in Cincinnati, because of possible neighborhood differences, comparisons of BAL SP with each of the reference groups seems appropriate. We used Round 3, the last preabatement blood lead sample time, as the basis for comparison even though the measurements were made in February 1990, about 6 months before the soil abatement, about 11 months before the first postabatement blood lead at Round 4 in 1991, and about 19 months before the Sept. 1991 blood lead sample at Round 6. It was not clear which pre-post comparisons were more appropriate, Round 3 versus Round 4 or Round 6. Both are reported here. Table 5-16 shows the results of the Round 3 versus Round 4 and Round 6 comparison of group BAL SP versus the two controls. None of the

TABLE 5-14. REPEATED MEASURES ANALYSIS OF VARIANCE FOR CINCINNATI STUDY: EFFECT OF TRUNCATION BETWEEN ROUNDS 4 AND 7

Study Group Abate Versus Control		Truncation Category			
		All	7-24 μg/dL	10-19 μg/dL	10-24 μg/L
Abate	Control	Reduction in Blood Lead (E_T) Age 9-17 Months[1,2]			
		(N=69)	(N=33)	(N=15)	
CIN NT (G)	CIN NT (M)	-4.56	NC	NC	
CIN SEI (P)	CIN NT(G)	1.06	-0.32	2.68	
CIN SEI (P)	CIN NT (M)	-3.50	NC	NC	
CIN SEI (P)	CIN I-SE (D)	-1.13	3.54	6.30	
CIN SEI (P)	CIN I-SE (F)	1.42	3.18	0.68	
		Reduction in Blood Lead (E_T) Age 18-41 Months[1,2]			
		(N=80)	(N=67)	(N=38)	(N=43)
CIN NT (G)	CIN NT (M)	-1.75	-1.05	2.42	0.06
CIN SEI (P)	CIN NT(G)	0.27	-0.48	0.75	-1.13
CIN SEI (P)	CIN NT (M)	-1.48	-1.53	-1.67	-1.07
CIN SEI (P)	CIN I-SE (D)	0.78	1.18	1.40	1.99
CIN SEI (P)	CIN I-SE (F)	0.23	0.39	-1.96	-0.49
		Reduction in Blood Lead (E_T) Age 42+ Months[1,2]			
		(N=70)	(N=47)	(N=31)	(N=36)
CIN NT (G)	CIN NT (M)		-2.86	-3.97	-4.00
CIN SEI (P)	CIN NT(G)		2.83	4.44	4.45
CIN SEI (P)	CIN NT (M)		-0.02	0.47	0.45
CIN SEI (P)	CIN I-SE (D)		1.18	1.75	1.81
CIN SEI (P)	CIN I-SE (F)		0.59	1.02	1.00

[1]Units are μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.

effect estimates for the large control group BAL P2 are statistically significant, nor is there any significant effect against BAL P1 using log blood lead, as shown in Table 5-16. The comparisons of BAL SP versus BAL P2 in Table 5-16 find a marginally significant difference (P = 0.16) between BAL SP and BAL P2 between Round 3 and Round 4, but the

TABLE 5-15. REPEATED MEASURES ANALYSIS OF VARIANCE FOR CINCINNATI STUDY: EFFECT OF TRUNCATION BETWEEN ROUNDS 4 AND 7

Study Group Abate Versus Control		Truncation Category			
		All	7-24 (μg/dL)	10-19 (μg/dL)	10-24 μg/L
Abate	Control	Reduction in Log Blood Lead (E_T) Age 9-17 Months[1,2]			
		(N=69)	(N=33)	(N=15)	
CIN NT (G)	CIN NT (M)		NC	NC	
CIN SEI (P)	CIN NT(G)		-0.179	0.251	
CIN SEI (P)	CIN NT (M)		NC	NC	
CIN SEI (P)	CIN I-SE (D)		0.123	0.561	
CIN SEI (P)	CIN I-SE (F)		-0.063	-0.075	
		Reduction in Log Blood Lead (E_T) Age 18-41 Months[1,2]			
		(N=80)	(N=67)	(N=38)	(N=43)
CIN NT (G)	CIN NT (M)	-0.188	-0.113	NC	0.052
CIN SEI (P)	CIN NT(G)	0.128	0.041	NC	-0.099
CIN SEI (P)	CIN NT (M)	-0.059	-0.072	NC	-0.047
CIN SEI (P)	CIN I-SE (D)	0.075	0.123	NC	0.183
CIN SEI (P)	CIN I-SE (F)	0.062	0.082	NC	-0.020
		Reduction in Log Blood Lead (E_T) Age 42+ Months[1,2]			
		(N=70)	(N=47)	(N=31)	(N=36)
CIN NT (G)	CIN NT (M)	-0.304^{M}	-0.292	-0.402	-0.403
CIN SEI (P)	CIN NT(G)	0.210^{M}	0.362	0.534	0.534
CIN SEI (P)	CIN NT (M)	-0.094	0.070	0.132	0.131
CIN SEI (P)	CIN I-SE (D)	0.123	0.272	0.382	0.363
CIN SEI (P)	CIN I-SE (F)	0.103	0.194	0.215	0.214

[1]Units are log μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.

effect is not statistically significant by Round 6. There is no significant effect after transforming to log blood lead, as shown in Table 5-16.

TABLE 5-16. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BALTIMORE STUDY: EFFECT OF AGE

Study Group		Age Group[1]			
		All Ages (N=463)	< 18 Months (N=16)	18-41 Months (N=88)	42+ Months (N=161)
Abate	Control	Reduction in Blood Lead (E_1) Between Rounds 3 and 4[1]			
BAL SP	BAL P1	0.07	5.70	0.22	0.06
BAL SP	BAL P2	1.77[M]	---	3.23	1.74[M]
		Reduction in Blood Lead (E_2) Between Rounds 3 and 6[1]			
BAL SP	BAL P1	-0.54	0.39	-0.44	-0.18
BAL SP	BAL P2	0.67[1T]	---	5.20[M]	0.55
		Reduction in Log Blood Lead (E_1) Between Rounds 3 and 4[2]			
BAL SP	BAL P1	-0.012	NC	NC	0.002
BAL SP	BAL P2	-0.002		NC	0.124
		Reduction in Log Blood Lead (E_2) Between Rounds 3 and 6[2]			
BAL SP	BAL P1	-0.013	NC	NC	NC
BAL SP	BAL P2	0.006	NC		

[1]Age is age in months at time of Round 1 blood sample.
[2]Units are μg/dL Pb in blood.
[3]Units are log μg/dL Pb in blood.

Age Effects

The design of the Baltimore study excluded most children younger than 18 months, and no significant effects were found in the few analyses that could be carried out with only 16 children at most in the three groups. There were 88 children in the age group 18-41 months, with a marginally significant effect between BAL SP and BAL P2 from Round 3 to Round 6 (P = 0.17), as found in Table 5-16. There were 161 children in the oldest group, and one of the effects was marginally significant for ages 42+ months, BAL SP versus BAL P2 from Round 3 to Round 4 (P = 0.12).

Effects of Blood Lead Truncation

Results are shown for Round 3 versus Round 6 comparisons only. Because the range of the blood lead concentrations at Round 3 in the Baltimore study was not constrained by study design, truncation of blood lead to the interval 7 to 24 μg/dL or smaller resulted in a substantial reduction in sample size. At ages 18-41 months, the number of children dropped from 88 for the full sample to 64 children in the range 7 to 24 μg/dL, and to 32 children in

the range 10 to 19 μg/dL. As shown in Table 5-17, the effects of BAL SP versus BAL P1 were somewhat larger. A similar pattern was seen in the log blood lead analyses in Table 5-17.

TABLE 5-17. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BALTIMORE STUDY: EFFECT OF TRUNCATION BETWEEN ROUNDS 3 AND 4

		TRUNCATION CATEGORY[1,2,3]				
		ALL	10-19	10-24	7-19	7-24
		<18: (N=16)	<18: (N=2)	<18: (N=4)	<18: (N=5)	<18: (N=7)
		18-42: (N=88)	18-42: (N=32)	18-42: (N=42)	18-42: (N=54	18-42: (N=64)
STUDY GROUP		>42: (N=161)	>42: (N=47)	>42: (N=53)	>42: (N=110)	>42: (N=120)
ABATE	CONTROL	REDUCTION IN BLOOD LEAD (E_t) FOR AGE <18 MONTHS				
BAL SP	BAL P1	5.70				14.7
BAL SP	BAL P2					
		REDUCTION IN BLOOD LEAD (E_t) FOR AGE 18-41 MONTHS				
BAL SP	BAL P1	0.22	-1.54		-1.56	-0.52
BAL SP	BAL P2	3.23				
		REDUCTION IN BLOOD LEAD (E_t) FOR AGE 42+ MONTHS				
BAL SP	BAL P1	0.06	-0.43	-0.22	-0.17	0.12
BAL SP	BAL P2	1.74[M]			1.68	1.39
		REDUCTION IN LOG BLOOD LEAD (E_t) FOR AGE <18 MONTHS[4]				
BAL SP	BAL P1					
BAL SP	BAL P2					
		REDUCTION IN LOG BLOOD LEAD (E_t) FOR AGE 18-41 MONTHS				
BAL SP	BAL P1		-0.300			
BAL SP	BAL P2					
		REDUCTION IN LOG BLOOD LEAD (E_t) FOR AGE 42+ MONTHS				
BAL SP	BAL P1	0.002	0.098	0.087	0.046	-0.020
BAL SP	BAL P2	0.124			0.110	0.123

[1]Units are μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.
[3]No optimal solution for missing cells in table.
[4]Units are log μg/dL Pb in blood.

The blood lead truncation also reduced the sample size for older children, from 161 children in the full sample to 116 children in the range of 7 to 24 μg/dL at Round 3, and only 47 children in the range 10 to 19 μg/dL. The effect size for BAL SP versus BAL P1 was larger in the truncated data sets, but not even marginally significant for blood lead in the range 7 to 24 μg/dL (smaller P = 0.21). There was, however, a suggestion of a reduction in BAL SP compared to the Area 1 control group, BAL P2 for children age 18 months and older, and possibly even a larger benefit relative to BAL P1 in the children < 18 months.

5.5.4 Boston Repeated Measures Analysis of Covariance (ANCOVA)

Many of the important covariate effects of age, race/ethnicity, and gender could be assessed by stratifying the data set, but the possible changes in response associated with continuous variables such as lead in soil and dust required a more general approach. Some preliminary results suggested that there may be systematic differences in response to changes in environmental lead across different treatment groups. These findings were explored more systematically in the longitudinal structural equation models (SEM) discussed in Section 5-6. We will briefly report the more significant findings from the ANCOVA analyses. The analyses were all carried out using log-transformed soil lead concentrations, dust lead concentrations, or dust lead loadings, because of the highly skewed distributions of these environmental lead variables. We also used log-transformed blood leads.

The results of the Phase 1 analyses are shown in Tables 5-18, 5-19, and 5-20. The blood lead effect (E_T) for repeated measures ANCOVA is calculated in the same manner as discussed previously for RM ANOVA. The change in the dust or soil lead regression coefficient for RM ANCOVA may be calculated as

$$(B_{gl} - B_{gr}) - (B_{hl} - B_{hr}) \qquad (5\text{-}22)$$

Table 5-18 shows the RM ANCOVA for log dust lead concentration, stratified by age. There were some marginally significant differences in the relationship between blood lead and dust lead concentration. The relationship, which was initially quite flat in all groups at Round 1, became much sharper in all groups at Round 3. However, the increasing slope of the log blood lead versus log dust lead concentration grew much more strongly in the dust

TABLE 5-18. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD CONCENTRATION REDUCTION ON LOG BLOOD LEAD (E_T) BETWEEN ROUNDS 1 AND 3

Study Group Abate Versus Control		Age Group[1,2]			
		All Ages (N=142)	9-17 Months (N=17)	18-41 Months (N=97)	42+ Months (N=28)
BOS SPI	BOS P-S	0.199	0.717	0.951	-2.421
BOS PI-S	BOS P-S	1.330[M]	3.339	1.524[M]	-3.622
BOS SPI	BOS PI-S	-1.131	-2.622	-0.573	1.201
Covariate: Log Dust Pb Concentration[3]					
BOS SPI	BOS P-S	-0.008	-0.095	-0.102	0.342
BOS PI-S	BOS P-S	-0.180[M]	-0.493	-0.199[M]	0.507
BOS SPI	BOS PI-S	0.173[M]	0.398	0.097	-0.165

[1]Units are log μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.
[3]Units are log μg/dL Pb in blood per log μg/g Pb in dust.

abatement group BOS PI-S between Round 1 and Round 3 than in either the soil abatement group BOS SPI or the control group BOS P-S. The change was most significant in the largest group, 18-41 months of age, but qualitatively similar in younger children. When log dust lead loading was used as a covariate, there were virtually no significant differences in blood lead response among treatment groups except for the youngest group, ages 9-17 months, where group BOS PI-S showed a relatively much more significant increase in the slope of the relationship ($P = 0.095$) than did BOS SPI or BOS P-S in Table 5-19. There were no significant effects on the relationship when log soil lead was used as a covariate. When both dust lead and soil lead were used as covariates, the effect sizes for slope were essentially those of the dust lead model, as shown in Table 5-20, with soil lead effects adding little to the predictive power of the model.

The results of the Phase 2 analyses are shown in Tables 5-20 and 5-21. Table 5-20 shows the RM ANCOVA for log dust lead concentration, stratified by age. There were some highly significant differences among treatment groups in the relationship between blood lead and dust lead concentration. The relationship of log blood lead versus log dust lead

TABLE 5-19. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD AND SOIL LEAD CONCENTRATION ON REDUCTION IN LOG BLOOD LEAD (E_T) BETWEEN ROUNDS 1 AND 3

Study Group Abate Versus Control		Age Group[1,2] All Ages (N=142)	9-17 Months (N=17)	18-41 Months (N=97)	42+ Months (N=28)
BOS SPI	BOS P-S			1.866[M]	-2.013
BOS PI-S	BOS P-S			0.981	-5.755
BOS SPI	BOS PI-S			0.884	3.742
Covariate: Log Dust Lead Concentration[3]					
BOS SPI	BOS P-S			-0.096	0.416
BOS PI-S	BOS P-S			-0.206[M]	0.438
BOS SPI	BOS PI-S			0.111	-0.022
Covariate: Log Soil Lead Concentration[4]					
BOS SPI	BOS P-S			-0.103	-0.151
BOS PI-S	BOS P-S			0.076	0.362
BOS SPI	BOS PI-S			-0.178	-0.513

[1]Units are log μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.
[3]Units are log μg/dL Pb in blood per μg/g Pb in dust.
[4]Units are log μg/dL Pb in blood per log μg/g Pb in soil.

concentration flattened much more strongly in the Phase 2 soil abatement group BOS PI-S between Round 3 and Round 4 than in either the soil abatement group BOS P-S or the Phase 2 non-abatement group BOS SPI. The change was most significant (P = 0.0019 for BOS PI-S versus BOS P-S, P = 0.029 for BOS PI-S versus BOS SPI) in the largest group, 18-41 months of age, but not separately estimatable in younger children. When log dust lead loading was used as a covariate, there were similar significant differences in blood lead response among treatment groups in the two older age groups, where group BOS PI-S showed a relatively much more significant decreases in the slope of the relationship than did either BOS SPI or P in Table 5-21. For 18-41 month old children, the effects on log blood lead versus log dust lead loading shown in Table 5-21 had significance levels P = 0.017 for BOS PI-S versus BOS SPI and P = 0.033 for BOS PI-S versus BOS P-S respectively. There were no significant effects on the relationship when log soil lead was used as a covariate.

TABLE 5-20. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD CONCENTRATION ON REDUCTION IN LOG BLOOD LEAD (E_r) BETWEEN ROUNDS 3 AND 4

Study Group Abate Versus Control		Age Group[1,2] All Ages (N=142)	9-17 Months (N=17)	18-41 Months (N=97)	42+ Months (N=28)
BOS SPI	BOS P-S	0.731	-6.685	2.828[M]	-3.720
BOS PI-S	BOS P-S	-3.575[S1]	---	-4.123[S1]	0.077
BOS SPI	BOS PI-S	-4.306[S1]	---	-6.950[S2]	4.797
Covariate: Log Dust Lead Concentration[3]					
BOS SPI	BOS P-S	-0.098	1.035	-0.432?	0.582
BOS PI-S	BOS P-S	0.558[S1]	---	0.628?	-0.101
BOS SPI	BOS PI-S	0.656[S1]	---	1.060?	-0.683

[1]Units are log μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.
[3]Units are μg/dL Pb in blood per unit logarithm difference in dust lead concentration. Equivalent to decimal percent change (see Equation 5-20).

TABLE 5-21. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD LOADING ON REDUCTION IN BLOOD LEAD (E_r) BETWEEN ROUNDS 3 AND 4

Study Group Abate Versus Control		Age Group[1,2] All Ages (N=128)	9-17 Months (N=15)	18-41 Months (N=89)	42+ Months (N=24)
BOS SPI	BOS P-S	0.120	NC	-0.393	0.764
BOS PI-S	BOS P-S	-0.607	NC	-1.289[1T]	2.178
BOS SPI	BOS PI-S	-0.727[M]	NC	-0.896[M]	-2.943[M]
Covariate: Log Dust Lead Loading					
BOS SPI	BOS P-S	-0.013	NC	0.099	0.106
BOS PI-S	BOS P-S	0.267[1T]	NC	0.459[S1]	0.670[M]
BOS SPI	BOS PI-S	0.280[1T]	NC	0.360[S1]	0.776[M]

[1]Units are log μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.
[3]Units are μg/dL Pb in blood per unit logarithm difference in dust lead concentration. Equivalent to decimal percent change (see Equation 5-20).

When both dust lead and soil lead were used as covariates, the effect sizes for slope were essentially those of the dust lead model, with soil lead effects adding little to the predictive power of the model.

When stratified analyses by race were carried out, the effects on dust lead slopes were larger and more significant among Afro-American children than for the sample as a whole. Table 5-22 is analogous to Table 5-18. The effect on dust lead slope for BOS PI-S versus BOS P-S between round 1 and round 3 is large, -0.528, and is highly significant ($P = 0.0078$). The effect on slope between BOS SPI and BOS PI-S is also large 0.386, and is nearly significant ($P = 0.058$). The significant whole-sample dust lead slope effects reflect the significant effects in the 18-41 month age group. However, slope effects for Afro-American children shown in Table 5-23 are nearly all non-significant when log dust lead loading is used as a covariate. Soil lead concentration is also not predictive for these children. When both log dust lead concentration and soil lead concentration are used as covariates, as shown in Table 5-24, there are large and highly significant differences in Phase 1 dust lead slope effects in group BOS PI-S, and large marginally significant soil lead slope effects as well, although these may reflect the collinearity between soil lead and dust lead in group BOS PI-S.

Stratified analyses for Phase 2 effects in Afro-American children are shown in Tables 5-25 and 5-26. Table 5-25 shows a large decrease in slope in group BOS PI-S, just as in Table 5-20, with $P = 0.0349$ for BOS PI-S versus BOS SPI in children of ages 18-41 months. However, in Table 5-26, slope effects for log dust lead loading are relatively large and significant, $P = 0.015$ for BOS PI-S versus BOS SPI and $P = 0.071$ for BOS PI-S versus BOS P-S in 18-41 month old Afro-American children.

In summary, these analyses suggest that there were some fairly substantial differences in the relationship between blood lead and dust lead during successive phases of the Boston study. The relationship between blood lead and dust lead was very flat in Round 1, and suggests that there may have been an attenuation in the real relationship due to selection or recruitment effects. After the Phase 1 abatement, the relationship became much more evident in the group BOS PI-S that received dust abatement, but not soil abatement in Phase 1 of the study. When group BOS PI-S received soil abatement during Phase 2 of the Boston study, much of the apparent relationship seems to have been attenuated or reversed.

TABLE 5-22. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD CONCENTRATION ON REDUCTION IN LOG BLOOD LEAD (E_T) BETWEEN ROUNDS 1 AND 3 FOR AFRO-AMERICAN CHILDREN

Study Group Abate Versus Control		Age Group[1,2] All Ages (N=71)	9-17 Months (N=11)	18-41 Months (N=44)	42+ Months (N=16)
BOS SPI	BOS P-S	0.549	NC	1.223	-3.269
BOS PI-S	BOS P-S	3.008[S1]	NC	3.865[S2]	-6.539
BOS SPI	BOS PI-S	-2.460[1T]	NC	-2.642[1T]	3.270
Covariate: Log Dust Lead Concentration[3]					
BOS SPI	BOS P-S	-0.056	NC	-0.142	0.448
BOS PI-S	BOS P-S	-0.413[S1]	NC	-0.528[S2]	0.872
BOS SPI	BOS PI-S	0.357[1T]	NC	0.386[1T]	-0.423

[1]Units are log μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.
[3]Units are μg/dL Pb in blood per unit logarithm difference in dust lead concentration. Equivalent to decimal percent change (see Equation 5-20).

TABLE 5-23. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD LOAD ON REDUCTION IN LOG BLOOD LEAD (E_T) BETWEEN ROUNDS 1 AND 3 FOR AFRO-AMERICAN CHILDREN

Study Group Abate Versus Control		Age Group[1,2] All Ages (N=71)	9-17 Months (N=11)	18-41 Months (N=44)	42+ Months (N=16)
BOS SPI	BOS P-S	0.109	NC	0.366	5.074
BOS PI-S	BOS P-S	0.439	NC	0.820*	0.597
BOS SPI	BOS PI-S	-0.330	NC	-0.453	4.478
Covariate: Log Dust Lead Load[3]					
BOS SPI	BOS P-S	0.015	NC	-0.035	-1.261
BOS PI-S	BOS P-S	-0.096	NC	-0.153+	-0.222
BOS SPI	BOS PI-S	0.111	NC	0.118	-1.038

[1]Units are log μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.
[3]Units are μg/dL Pb in blood per unit logarithm difference in dust lead concentration. Equivalent to decimal percent change (see Equation 5-20).

TABLE 5-24. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD CONCENTRATION, SOIL LEAD CONCENTRATION ON REDUCTION IN LOG BLOOD LEAD (E_T) BETWEEN ROUNDS 1 AND 3 FOR AFRO-AMERICAN CHILDREN

Study Group Abate Versus Control		Age Group[1,2] All Ages (N=71)	9-17 Months (N=11)	18-41 Months (N=44)	42+ Months (N=16)
BOS SPI	BOS P-S	0.609	NC	1.672	NC
BOS PI-S	BOS P-S	1.916	NC	1.326	NC
BOS SPI	BOS PI-S	-1.307	NC	0.346	NC
Covariate: Log Dust Lead Concentration[3]					
BOS SPI	BOS P-S	-0.066	NC	-0.169	NC
BOS PI-S	BOS P-S	-0.436[S1]	NC	-0.574[S2]	NC
BOS SPI	BOS PI-S	0.370[1T]	NC	0.405[S1]	NC
Covariate: Log Soil Lead Concentration[4]					
BOS SPI	BOS P-S	0.030	NC	0.003	NC
BOS PI-S	BOS P-S	0.163	NC	0.370[M]	NC
BOS SPI	BOS PI-S	-0.132	NC	-0.367[M]	NC

[1]Units are log μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.
[3]Units are μg/dL Pb in blood per unit logarithm difference in dust lead concentration. Equivalent to decimal percent change (see Equation 5-20).
[4]Units are μg/dL Pb in blood per unit logarithm difference in soil lead concentration. Equivalent to decimal percent change (see Equation 5-20).

Additional analyses using SEM to further evaluate the changing patterns in the soil lead - dust lead - blood lead pathways in different treatment groups seemed to be useful in understanding some of these effects.

5.5.5 Cincinnati Repeated Measures Analysis of Covariance (ANCOVA)

The repeated measures analyses for Cincinnati were directed towards assessing the role of longitudinal group differences associated with different dust indices. The basic Cincinnati model was run with each of the logarithms of floor dust concentration and loading, entry dust concentration and loading, window dust concentration and loading. The results are shown in Table 5-27.

TABLE 5-25. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD LOADING ON REDUCTION IN LOG BLOOD LEAD (E_τ) BETWEEN ROUNDS 3 AND 4 FOR AFRO-AMERICAN CHILDREN

Study Group Abate Versus Control		Age Group[1,2] All Ages (N=64)	9-17 Months (N=8)	18-41 Months (N=40)	42+ Months (N=16)
BOS SPI	BOS P-S	0.324	NC	-0.683	0.961
BOS PI-S	BOS P-S	-0.721	NC	-2.045[S1]	-3.000[M]
BOS SPI	BOS PI-S	-1.045	NC	-1.362[M]	-3.961[M]
Covariate: Log Dust Lead Loading					
BOS SPI	BOS P-S	-0.042	NC	0.214	-0.128[M]
BOS PI-S	BOS P-S	0.316[M]	NC	0.692[S1]	0.941[M]
BOS SPI	BOS PI-S	0.357[M]	NC	0.478[1T]	1.069[1T]

[1]Units are log μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.
[3]Units are reduction of natural logarithm μg/dL Pb in blood per unit logarithm difference in dust lead concentration. Equivalent to decimal percent change (see Equation 5-20).

TABLE 5-26. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD CONCENTRATION ON REDUCTION IN LOG BLOOD LEAD (E_τ) BETWEEN ROUNDS 3 AND 4 FOR AFRO-AMERICAN CHILDREN

Study Group Abate Versus Control		Age Group[1,2] All Ages (N=64)	9-17 Months (N=8)	18-41 Months (N=40)	42+ Months (N=16)
BOS SPI	BOS P-S	0.642	NC	0.340	0.150
BOS PI-S	BOS P-S	-3.629[M]	NC	-5.575[S1]	6.796
BOS SPI	BOS PI-S	-4.271[M]	NC	-5.915[M]	6.646
Covariate: Log Dust Lead Concentration[3]					
BOS SPI	BOS P-S	-0.050	NC	-0.034	0.063
BOS PI-S	BOS P-S	0.588[1T]	NC	0.863[S1]	-0.886
BOS SPI	BOS PI-S	0.639[M]	NC	0.897[S1]	-0.949

[1]Units are log μg/dL Pb in blood.
[2]Age is age in months at time of Round 1 blood sample.
[3]Units are reduction of natural logarithm μg/dL Pb in blood per unit logarithm difference in dust lead concentration. Equivalent to decimal percent change (see Equation 5-20).

TABLE 5-27. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR CINCINNATI STUDY: REDUCTION IN BLOOD LEAD (E_r) BETWEEN ROUNDS 1 AND 4

Study Group		Log Floor Dust Concentration	Log Entry Dust Concentration	Log Window Dust Concentration	Log Floor Dust Pb Loading	Log Entry Dust Pb Loading	Log Window Dust Pb Loading
Abate Versus Control				Intercept Effect[1]			
CIN NT(G)	CIN NT(M)	0.539	2.208	-3.362[IT]	1.605[M]	1.417[SI]	-0.200
CIN SEI (P)	CIN NT(G)	0.429	-0.467	0.078	-0.188	-0.755[IT]	-0.775[M]
CIN SEI (P)	CIN NT(M)	0.969	1.741	-3.284[M]	1.417[M]	0.662	-0.975
CIN SEI (P)	CIN I-SE(D)	0.949	-0.283	-1.494	0.355	-1.095[IT]	-1.846[SI]
CIN SEI (P)	CIN I-SE(F)	0.416	-0.593	-0.130	-0.701	-0.528	-0.337
				Covariate Effect[2]			
CIN NT(G)	CIN NT(M)	0.015	-0.244	0.540[SI]	-0.277	-0.134	0.102
CIN SEI (P)	CIN NT(G)	-0.134	0.010	-0.055	-0.028	0.077	0.055
CIN SEI (P)	CIN NT(M)	-0.118	-0.234	0.486[IT]	-0.305	-0.057	0.158
CIN SEI (P)	CIN I-SE(D)	-0.189	-0.001	0.187	-0.100	0.138[M]	0.195[SI]
CIN SEI (P)	CIN I-SE(F)	-0.092	0.067	0.006	0.112	0.060	0.032

[1]Units are log μg/dL Pb in blood.
[2]Units are reduction of natural logarithm μg/dL Pb in blood per unit logarithm difference in dust lead concentration. Equivalent to decimal percent change (see Equation 5-20).

There was no indication of any strong change in the response of log blood lead to floor dust or entry dust during the study. There was, however, an indication that the relationship of blood lead to window dust changed between some of the neighborhoods during Phase 1 of the study. Table 5-27 shows a statistically significant difference in change in log window dust concentration regression coefficients between CIN NT(G) and CIN NT(M) ($P = 0.0496$) and a nearly significant difference between CIN SEI(P) and CIN NT(M) ($P = 0.08$), while there was almost no difference in the longitudinal change between CIN SEI(P) and CIN NT(G) responses to window dust lead ($P = 0.95$). This suggests that CIN NT(M) was a neighborhood in which some substantial external change occurred that was manifested as a difference in the response to window dust. There was also a significant effect in covariate response for window dust lead loading in CIN SEI(P) vs CIN I-SE(D) ($P = 0.025$), which was not manifested as strongly as a change in response to window dust lead concentration; perhaps changes in window dust loadings were an important factor. The window dust covariate effects that were significant and positive were associated with negative changes in

the intercept for the window dust lead concentration model, P = 0.095 for CIN NT(G) vs CIN NT(M) and P = 0.11 for CIN SEI(P) vs CIN NT(M), and in the intercept for the window dust lead loading model, P = 0.014 for CIN SEI(P) vs. CIN I-SE(D).

There were some significant differences in the intercept model where the log entry dust lead loading was used as a covariate, between CIN NT(G) and CIN NT(M) (P = 0.0494), CIN SEI(P) and CIN NT(G) (P = 0.08), and between CIN SEI(P) and CIN I-SE(D) (P = 0.06). The only covariate response change of even marginal significance was for CIN SEI(P) vs CIN I-SE(D) (P = 0.11). The lack of any significant effects of any sort when log of entry dust concentration was used as a covariate suggests that changes in entry dust loading may have occured in several of these neighborhoods.

These analyses suggested that changes in response to window dust lead may have played a role in blood leads among the Cincinnati neighborhoods. A more detailed evaluation of some dust lead pathway models using LSEM showed some modest indications of changes in the pathway components from window dust to floor dust.

5.5.6 Baltimore Repeated Measures Analysis of Covariance (ANCOVA)

We have not carried out either repeated measures ANCOVA models or structural equation models for the Baltimore study because of the limited environmental data in the Baltimore study. The results of the Boston and Cincinnati studies have shown that there can be substantial changes in dust lead concentrations from one round to another, both in abated and non-abated residences. Since there are strong and statistically significant relationships between blood lead and current dust lead measurements in all rounds in the Cincinnati study and in all rounds after Round 1 in Boston, it appears necessary to have dust lead measurements that reasonably characterize each round of blood lead measurements. However, the Baltimore study did not collect any post-abatement dust lead measurements for non-abated residences, nor long-term post-abatement dust lead measurements in abated residences. The often large intervals between pre-abatement dust lead measurements and Round 3 blood lead measurements may not even provide adequate information about baseline exposures. While soil lead concentrations in non-abated residences appear to change very slowly over time, some post-abatement soil lead concentrations may increase because of recontamination, and additional post-abatement soil lead data in both abated and non-abated

residences would have been desirable. In earlier drafts of this report, EPA evaluated several models in which the dust lead and soil lead post-abatement data that were not available were imputed by assuming that the post-abatement environmental measurements were equal to the pre-abatement measurements. Based on these earlier assessments and on reviewer comments, we conclude that this approach does not provide adequate information about actual post-abatement environmental exposures. Therefore, we were unable to use these data for time-varying covariate adjustment models for the Baltimore study. Repeated measures ANOVA models were stratified for time-constant covariates such as age and gender, and our conclusions are based on these analyses.

5.6 COMPARISON USING LONGITUDINAL STRUCTURAL EQUATION MODELS

5.6.1 Boston Study Longitudinal Structural Equation Models

Recall from Section 5.1.1.2 that the equations for the longitudinal structural equation model are

$$Y_{ir} = G_{gr} + X_{ir}B_{gr} + Z_{ir}F_{gr} + Y_{ir-1}A_j + e_{ir} \quad (5\text{-}18)$$

$$X_{ir} = C_{gr} + Z_{ir}D_{gr} + Z_{ir-1}K_{gr} + W_{ir-1}L_{gr} + d_{ir} \quad (5\text{-}19)$$

and that the model adjusts for the simultaneous fitting of multiple relationships. In a scheme of thirty-two models run by the SAS PROC MIXED procedure, shown on Table 5-28, each model was run with a component of the structural equation, either a common intercept term (G_{gi} or C_{gi}) or a pathway regression coefficient (F_{gj}, B_{gj}, L_{gj}, or D_{gi}), for Round 3 blood lead or Round 3 dust lead. In some cases, these coefficients were separated into three intercept terms or three regression coefficients, one for each of the three treatment groups BOS SPI, BOS PI-S, or BOS P-S. The best-fitting of the 32 models, were models 1, 2, 10, 11, 17, and 30, and these were used for the reported output in Tables 5-29 through 5-30. The longitudinal structural equation models allow effects adjusted for changes in both concentration (X_{gr} in Group g at Round r) and regression coefficients,

TABLE 5-28. MODELS FOR TREATMENT GROUP EFFECTS IN BOSTON LONGITUDINAL STRUCTURAL EQUATION MODELS

MODEL NUMBER	BLOOD LEAD INTERCEPT	SOIL-BLOOD COEFFICIENT	DUST-BLOOD COEFFICIENT	SOIL-DUST MODEL	WINDOW-DUST MODEL
1	G	F	B	C, D	C, L
2	G	F_{SPI}, F_{PI-S}, F_{P-S}	B	C, D	C, L
3	G	F_{SPI}, F_{PI-S}, F_{P-S}	B_{SPI}, B_{PI-S}, B_{P-S}	C, D	C, L
4	G	F_{SPI}, F_{PI-S}, F_{P-S}	B_{SPI}, B_{PI-S}, B_{P-S}	C_{SPI}, C_{PI-S}, C_{P-S} D_{SPI}, D_{PI-S}, D_{P-S}	C, L
5	G	F_{SPI}, F_{PI-S}, F_{P-S}	B_{SPI}, B_{PI-S}, B_{P-S}	C_{SPI}, C_{PI-S}, C_{P-S} D_{SPI}, D_{PI-S}, D_{P-S}	C_{SPI}, C_{PI-S}, C_{P-S} L_{SPI}, L_{PI-S}, L_{P-S}
6	G	F_{SPI}, F_{PI-S}, F_{P-S}	B_{SPI}, B_{PI-S}, B_{P-S}	C, D	C_{SPI}, C_{PI-S}, C_{P-S} L_{SPI}, L_{PI-S}, L_{P-S}
7	G	F_{SPI}, F_{PI-S}, F_{P-S}	B	C_{SPI}, C_{PI-S}, C_{P-S} D_{SPI}, D_{PI-S}, D_{P-S}	C, L
8	G	F_{SPI}, F_{PI-S}, F_{P-S}	B	C_{SPI}, C_{PI-S}, C_{P-S} D_{SPI}, D_{PI-S}, D_{P-S}	C_{SPI}, C_{PI-S}, C_{P-S} L_{SPI}, L_{PI-S}, L_{P-S}
9	G	F_{SPI}, F_{PI-S}, F_{P-S}	B	C, D	C_{SPI}, C_{PI-S}, C_{P-S} L_{SPI}, L_{PI-S}, L_{P-S}
10	G	F	B_{SPI}, B_{PI-S}, B_{P-S}	C, D	C, L
11	G	F	B_{SPI}, B_{PI-S}, B_{P-S}	C_{SPI}, C_{PI-S}, C_{P-S} D_{SPI}, D_{PI-S}, D_{P-S}	C, L
12	G	F	B_{SPI}, B_{PI-S}, B_{P-S}	C_{SPI}, C_{PI-S}, C_{P-S} D_{SPI}, D_{PI-S}, D_{P-S}	C_{SPI}, C_{PI-S}, C_{P-S} L_{SPI}, L_{PI-S}, L_{P-S}
13	G	F	B_{SPI}, B_{PI-S}, B_{P-S}	C, D	C_{SPI}, C_{PI-S}, C_{P-S} L_{SPI}, L_{PI-S}, L_{P-S}
14	G	F	B	C_{SPI}, C_{PI-S}, C_{P-S} D_{SPI}, D_{PI-S}, D_{P-S}	C, L
15	G	F	B	C_{SPI}, C_{PI-S}, C_{P-S} D_{SPI}, D_{PI-S}, D_{P-S}	C_{SPI}, C_{PI-S}, C_{P-S} L_{SPI}, L_{PI-S}, L_{P-S}
16	G	F	B	C, D	C_{SPI}, C_{PI-S}, C_{P-S} L_{SPI}, L_{PI-S}, L_{P-S}
17	G_{SPI}, G_{PI-S}, G_{P-S}	F	B	C, D	C, L
18	G_{SPI}, G_{PI-S}, G_{P-S}	F_{SPI}, F_{PI-S}, F_{P-S}	B	C, D	C, L
19	G_{SPI}, G_{PI-S}, G_{P-S}	F_{SPI}, F_{PI-S}, F_{P-S}	B_{SPI}, B_{PI-S}, B_{P-S}	C, D	C, L
20	G_{SPI}, G_{PI-S}, G_{P-S}	F_{SPI}, F_{PI-S}, F_{P-S}	B_{SPI}, B_{PI-S}, B_{P-S}	C_{SPI}, C_{PI-S}, C_{P-S} D_{SPI}, D_{PI-S}, D_{P-S}	C, L
21	G_{SPI}, G_{PI-S}, G_{P-S}	F_{SPI}, F_{PI-S}, F_{P-S}	B_{SPI}, B_{PI-S}, B_{P-S}	C_{SPI}, C_{PI-S}, C_{P-S} D_{SPI}, D_{PI-S}, D_{P-S}	C_{SPI}, C_{PI-S}, C_{P-S} L_{SPI}, L_{PI-S}, L_{P-S}
22	G_{SPI}, G_{PI-S}, G_{P-S}	F_{SPI}, F_{PI-S}, F_{P-S}	B_{SPI}, B_{PI-S}, B_{P-S}	C, D	C_{SPI}, C_{PI-S}, C_{P-S} L_{SPI}, L_{PI-S}, L_{P-S}
23	G_{SPI}, G_{PI-S}, G_{P-S}	F_{SPI}, F_{PI-S}, F_{P-S}	B	C_{SPI}, C_{PI-S}, C_{P-S} D_{SPI}, D_{PI-S}, D_{P-S}	C, L

TABLE 5-28 (cont'd). MODELS FOR TREATMENT GROUP EFFECTS IN BOSTON LONGITUDINAL STRUCTURAL EQUATION MODELS

MODEL NUMBER	BLOOD LEAD INTERCEPT	SOIL-BLOOD COEFFICIENT	DUST-BLOOD COEFFICIENT	SOIL-DUST MODEL	WINDOW-DUST MODEL
24	G_{SPI}, $G_{PI\text{-}S}$, $G_{P\text{-}S}$	F_{SPI}, $F_{PI\text{-}S}$, $F_{P\text{-}S}$	B	C_{SPI}, $C_{PI\text{-}S}$, $C_{P\text{-}S}$ D_{SPI}, $D_{PI\text{-}S}$, $D_{P\text{-}S}$	C_{SPI}, $C_{PI\text{-}S}$, $C_{P\text{-}S}$ L_{SPI}, $L_{PI\text{-}S}$, $L_{P\text{-}S}$
25	G_{SPI}, $G_{PI\text{-}S}$, $G_{P\text{-}S}$	F_{SPI}, $F_{PI\text{-}S}$, $F_{P\text{-}S}$	B	C, D	C_{SPI}, $C_{PI\text{-}S}$, $C_{P\text{-}S}$ L_{SPI}, $L_{PI\text{-}S}$, $L_{P\text{-}S}$
26	G_{SPI}, $G_{PI\text{-}S}$, $G_{P\text{-}S}$	F	B_{SPI}, $B_{PI\text{-}S}$, $B_{P\text{-}S}$	C, D	C, L
27	G_{SPI}, $G_{PI\text{-}S}$, $G_{P\text{-}S}$	F	B_{SPI}, $B_{PI\text{-}S}$, $B_{P\text{-}S}$	C_{SPI}, $C_{PI\text{-}S}$, $C_{P\text{-}S}$ D_{SPI}, $D_{PI\text{-}S}$, $D_{P\text{-}S}$	C, L
28	G_{SPI}, $G_{PI\text{-}S}$, $G_{P\text{-}S}$	F	B_{SPI}, $B_{PI\text{-}S}$, $B_{P\text{-}S}$	C_{SPI}, $C_{PI\text{-}S}$, $C_{P\text{-}S}$ D_{SPI}, $D_{PI\text{-}S}$, $D_{P\text{-}S}$	C_{SPI}, $C_{PI\text{-}S}$, $C_{P\text{-}S}$ L_{SPI}, $L_{PI\text{-}S}$, $L_{P\text{-}S}$
29	G_{SPI}, $G_{PI\text{-}S}$, $G_{P\text{-}S}$	F	B_{SPI}, $B_{PI\text{-}S}$, $B_{P\text{-}S}$	C, D	C_{SPI}, $C_{PI\text{-}S}$, $C_{P\text{-}S}$ L_{SPI}, $L_{PI\text{-}S}$, $L_{P\text{-}S}$
30	G_{SPI}, $G_{PI\text{-}S}$, $G_{P\text{-}S}$	F	B	C_{SPI}, $C_{PI\text{-}S}$, $C_{P\text{-}S}$ D_{SPI}, $D_{PI\text{-}S}$, $D_{P\text{-}S}$	C, L
31	G_{SPI}, $G_{PI\text{-}S}$, $G_{P\text{-}S}$	F	B	C_{SPI}, $C_{PI\text{-}S}$, $C_{P\text{-}S}$ D_{SPI}, $D_{PI\text{-}S}$, $D_{P\text{-}S}$	C_{SPI}, $C_{PI\text{-}S}$, $C_{P\text{-}S}$ L_{SPI}, $L_{PI\text{-}S}$, $L_{P\text{-}S}$
32	G_{SPI}, $G_{PI\text{-}S}$, $G_{P\text{-}S}$	F	B	C, D	C_{SPI}, $C_{PI\text{-}S}$, $C_{P\text{-}S}$ L_{SPI}, $L_{PI\text{-}S}$, $L_{P\text{-}S}$

TABLE 5-29. LONGITUDINAL STRUCTURAL EQUATION MODELS MODEL ASSESSMENT STATISTICS IN BOSTON STUDY USING ESTIMATED BLOOD LEAD PERSISTENCE FACTOR

		MODEL ASSESSMENT STATISTICS				
Response Variable	Statistic	MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17
Log Blood Lead RD3	RMSE	0.30089	0.30049	0.29796	0.29797	0.29696
Log Blood Lead RD1	RMSE	0.37416	0.36512	0.33282	0.33284	0.38106
Log Dust Lead Conc. RD3	RMSE	0.62147	0.62081	0.60797	0.62998	0.61399
Log Dust Lead Conc. RD1	RMSE	0.79374	0.79092	0.78507	0.78513	0.78683
All	N*OBJ	42.75	41.86	12.43	10.95	43.45

[1]RMSE = Root mean squared error.

TABLE 5-30. LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: REGRESSION COEFFICIENTS USING ESTIMATED BLOOD LEAD PERSISTENCE FACTOR

Predictor Variable		MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17
		RESPONSE VARIABLE BLOOD LEAD ROUND 3				
Intercept[1]	ALL GROUPS	1.96	2.04	2.42	2.42	
	BOS SPI					1.95
	BOS PI-S					3.28
	BOS P-S					4.41
Soil Pb Round 3[2]	ALL GROUPS	0.719		0.032	0.032	0.043
	BOS SPI		0.511			
	BOS PI-S		0.680			
	BOS P-S		0.817			
Dust Pb Conc. Round 3[2]	ALL GROUPS	0.318	0.302			0.429
	BOS SPI			0.182	0.180	
	BOS PI-S			1.275	1.274	
	BOS P-S			1.343	1.342	
Blood Lead Round 1[3]		0.6088[1T]	0.6060[M]	0.5890	0.5890	0.5931
		RESPONSE VARIABLE: BLOOD LEAD ROUND 1				
Intercept[1]		10.75[S4]	11.11[S4]	11.21[S4]	11.22[S4]	10.63[S4]
Soil Pb Round 1[2]		-0.291[M]	-0.293[M]	0.151	0.149	-0.316[M]
Dust Pb Round 1[2]		0.524[S1]	0.449[S1]	0.038	0.038	0.574[S1]
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1				
Intercept[4]		1826[S4]	1736[S4]	1408[S2]	1414[S2]	1256[S3]
Soil Pb Round 1[5]		-0.204[M]	-0.174[M]	-0.073	-0.074	-0.010
Window Dust Pb Round 1[5]		0.0684[S4]	0.0693[S4]	0.0714[S4]	0.0716[S4]	0.0608[S4]
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 3				
Intercept[4]	ALL GROUPS	791[S4]	808[S4]	675[S4]		834[S4]
	BOS SPI				849[S4]	
	BOS PI-S				358	
	BOS P-S				130	

TABLE 5-30 (cont'd). LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: REGRESSION COEFFICIENTS USING ESTIMATED BLOOD LEAD PERSISTENCE FACTOR

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17
Soil Pb Conc. Round 3[5]	ALL GROUPS	0.201[S4]	0.198[S4]	0.198[S3]		0.189[S3]
	BOS SPI				-0.062	
	BOS PI-S				0.303	
	BOS P-S				0.473	
Window Dust Pb Conc. Round 3[5]		0.0111[S2]	0.0103[S1]	0.0092[S2]	0.0074[S1]	0.0067[1T]

[1]Units are μg/dL Pb in blood.
[2]Units are μg/dL Pb in blood per 1000 μg/g Pb in dust or soil.
[3]Units are μg/dL Pb in blood at Round 3 per μg/dL Pb in blood at Round 1.
[4]Units are μg/g Pb in dust.
[5]Units are μg/g Pb in floor dust per μg/g Pb in soil or window dust.

TABLE 5-31. LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: MODEL ASSESSMENT STATISTICS USING FIXED BLOOD LEAD PERSISTENCE FACTOR

Response Variable	STATISTIC	MODEL ASSESSMENT STATISTICS MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30
Log Blood Pb Round 3	RMSE	0.2915	0.2945	0.3060	0.3102	0.3036	0.3120
Log Blood Pb Round 1	RMSE	0.3392	0.3393	0.3413	0.3411	0.3400	0.3402
Log Dust Pb Conc. Round 3	RMSE	0.6111	0.6110	0.6094	0.6412	0.6094	0.6283
Log Dust Pb Conc. Round 1	RMSE	0.7918	0.7921	0.7898	0.7916	0.7894	0.7908
All	N*OBJ	17.57	17.47	16.47	14.37	16.34	14.54

[1]RMSE = Root mean squared error.

TABLE 5-32. LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: REGRESSION COEFFICIENTS USING FIXED BLOOD LEAD PERSISTENCE FACTOR

Predictor Variable		REGRESSION COEFFICIENT					
		MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30
		RESPONSE VARIABLE: BLOOD LEAD ROUND 3					
Intercept[1]	ALL GROUPS	1.37[S1]	1.41[S1]	1.05[M]	1.27[M]		
	BOS SPI					0.24	-0.03
	BOS PI-S					1.80[S1]	2.51[S1]
	BOS P-S					2.10[S1]	2.50[S1]
Soil Pb Round 3[2]	ALL GROUPS	0.430[1T]		-0.202	-0.419	-0.263	-0.512
	BOS SPI		0.686				
	BOS PI-S		0.424[M]				
	BOS P-S		0.473[1T]				
Floor Dust Pb Conc. Roung 3[2]	ALL GROUPS	0.795[M]	0.736[M]			1.752[S1]	1.931[S1]
	BOS SPI			1.014[M]	0.820		
	BOS PI-S			2.101[1T]	2.569[S1]		
	BOS P-S			2.278[S1]	2.407[S1]		
Blood Pb Round 1[3]		0.589	0.589	0.589	0.589	0.589	0.589
		RESPONSE VARIABLE: BLOOD LEAD ROUND 1					
Intercept[1]		12.74[S4]	12.75[S4]	12.70[S4]	12.51[S4]	12.66[S4]	12.55[S4]
Soil Pb Round 1[2]		-0.200	-0.204	-0.143	-0.043	-0.150	-0.072
Dust Pb Round 1[2]		-0.029	-0.030	-0.063	-0.066	-0.050	-0.055
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1					
Intercept[4]		1701[S4]	1721[S4]	1513[S4]	1647[S4]	1521[S4]	1695[S4]
Soil Pb Round 1[5]		-0.175[M]	-0.180[M]	-0.130	-0.172[M]	-0.125	-0.170[M]
Window Dust Pb Round 1[5]		0.0678[S4]	0.0678[S4]	0.0683[S4]	0.0737[S4]	0.0667[S4]	0.0701[S4]

TABLE 5-32 (cont'd). LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: REGRESSION COEFFICIENTS USING FIXED BLOOD LEAD PERSISTENCE FACTOR

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 3					
Intercept[4]	ALL GROUPS	705[S4]	707[S4]	713[S4]		717[S4]	
	BOS SPI				788[S4]		787[S4]
	BOS PI-S				109		367
	BOS P-S				744		485
Soil Pb Conc. Round 3[5]	ALL GROUPS	0.222[S4]	0.221[S4]	0.209[S4]		0.214[S4]	
	BOS SPI				0.192		0.197
	BOS PI-S				0.392		0.294
	BOS P-S				0.242		0.347
Window Dust Pb Conc. Round 3[5]		0.0095[S2]	0.0094[S2]	0.0091[S2]	0.0082[S1]	0.0083[S1]	0.0076[S1]

[1]Units are μg/dL Pb in blood.
[2]Units are μg/dL Pb in blood per 1000 μg/g Pb in dust or soil.
[3]Units are μg/dL Pb in blood at Round 3 per μg/dL Pb in blood at Round 1.
[4]Units are μg/g Pb in dust.
[5]Units are μg/g Pb in floor dust per μg/g Pb in soil or window dust.

TABLE 5-33. LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: MODEL ASSESSMENT STATISTICS USING FIXED BLOOD LEAD PERSISTENCE FACTOR FOR MALES

Response Variable	Statistic	MODEL ASSESSMENT STATISTICS MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30
Log Blood Pb Round 3	RMSE	0.26227	0.26907	0.28664	0.26284	0.27831	0.26417
Log Blood Pb Round 1	RMSE	0.37303	0.37510	0.37003	0.35280	0.37329	0.35432
Log Dust Pb Conc. Round 3	RMSE	0.62102	0.61741	0.61503	0.61901	0.61665	0.61574
Log Dust Lead Conc. Round 1	RMSE	0.77072	0.76918	0.77163	0.78928	0.76631	0.78474
All	N*OBJ	19.09	19.20	19.08	13.28	19.05	12.89

[1]Units are reduction of μg/dL Pb in blood per reduction of 1000 μg/g Pb in soil.
[2]Age is age in months at time of Round 1 blood sample.

TABLE 5-34. LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: REGRESSION COEFFICIENTS USING FIXED BLOOD LEAD PERSISTENCE FACTOR FOR MALES

Predictor Variable		REGRESSION COEFFICIENT					
		MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30
		RESPONSE VARIABLE: BLOOD LEAD ROUND 3					
Intercept[1]	ALL GROUPS	2.20[S3]	1.99[S2]	1.58[1T]	2.37[S2]		
	BOS SPI					1.37[M]	1.74[1T]
	BOS PI-S					2.63[S1]	3.35[S2]
	BOS P-S					2.01[M]	2.88[S1]
Soil Pb Round 3[2]	ALL GROUPS	1.007[S4]		0.607	0.055	0.505	-0.050
	BOS SPI		0.757				
	BOS PI-S		1.193[S4]				
	BOS P-S		0.858[S1]				
Floor Dust Pb Conc. Round 3[2]	ALL GROUPS	0.350	0.509			1.185[M]	0.871
	BOS SPI			1.008[M]	0.340		
	BOS PI-S			1.476	1.401		
	BOS P-S			1.536[M]	0.945		
Blood Pb Round 1[3]		0.589	0.589	0.589	0.589	0.589	0.589
		RESPONSE VARIABLE: BLOOD LEAD ROUND 1					
Intercept[1]		14.46[S4]	14.56[S3]	14.21[S4]	12.64[S4]	14.39[S4]	12.64[S4]
Soil Pb Round 1[2]		-0.786[S2]	-0.850[S2]	-0.735[S1]	-0.244	-0.812[S2]	-0.283
Dust Pb Round 1[2]		0.016	0.054	0.034	0.115	0.054	0.137
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1					
Intercept[4]		1416[S4]	1458[S4]	1315[S4]	541[1T]	1410[S4]	582[S1]
Soil Pb Round 1[5]		-0.141	-0.150	-0.115	0.276[S1]	-0.125	0.270[S1]
Window Dust Pb Round 1[5]		0.0750[S4]	0.0750[S4]	0.0764[S4]	0.0510[S4]	0.0739[S4]	0.0514[S4]
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 3					
Intercept[4]	ALL GROUPS	895[S4]	890[S4]	881[S4]		892[S4]	
	BOS SPI				761[S4]		787[S4]
	BOS PI-S				729[S1]		696[1T]
	BOS P-S				-292		-323

TABLE 5-34 (cont'd). LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: REGRESSION COEFFICIENTS USING FIXED BLOOD LEAD PERSISTENCE FACTOR FOR MALES

Predictor Variable		REGRESSION COEFFICIENT					
		MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30
Soil Pb Conc. Round 3[5]	ALL GROUPS	0.223[S4]	0.235[S4]	0.232[S4]		0.247[S4]	
	BOS SPI				0.237		0.217
	BOS PI-S				0.018		0.046
	BOS P-S				0.918[S1]		0.930[S1]
Window Dust Pb Conc. Round 3[5]		-0.0021	-0.0017	-0.0012	0.0076[S1]	-0.0015	0.0068[S1]

[1]Units are μg/dL Pb in blood.
[2]Units are μg/dL Pb in blood per 1000 μg/g Pb in dust or soil.
[3]Units are μg/dL Pb in blood at Round 3 per μg/dL Pb in blood at Round 1.
[4]Units are μg/g Pb in dust.
[5]Units are μg/g Pb in floor dust per μg/g Pb in soil or window dust.

TABLE 5-35. LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: MODEL ASSESSMENT STATISTICS USING FIXED BLOOD LEAD PERSISTENCE FACTOR FOR FEMALES

ResponSe Variable	STATISTIC	MODEL ASSESSMENT STATISTICS					
		MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30
Log Blood Pb Round 3	RMSE[1]	0.31964	0.33652	0.31968	0.31755	0.35483	0.31914
Log Blood Pb Round 1	RMSE	0.34553	0.33859	0.32730	0.38193	0.32931	0.39155
Log Dust Pb Conc. Round 3	RMSE	0.70085	0.69726	0.69855	0.69354	0.69200	0.69940
Log Dust Pb Conc. Round 1	RMSE	0.89148	0.87654	0.85724	0.86649	0.85785	0.86949
All	N*OBJ	13.15	12.71	11.77	10.87	12.15	11.15

[1]RMSE = Root mean squared error.

TABLE 5-36. LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: REGRESSION COEFFICIENTS USING FIXED BLOOD LEAD PERSISTENCE FACTOR FOR FEMALES

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30
		RESPONSE VARIABLE: BLOOD LEAD ROUND 3					
Intercept[1]	ALL GROUPS	1.15[M]	1.87[S1]	1.42[1T]	0.14		
	BOS SPI					1.84[M]	-0.21
	BOS PI-S					2.75[S1]	0.64
	BOS P-S					3.97[S3]	0.77
Soil Pb Round 3[2]	ALL GROUPS	0.416[M]		0.090	0.080	0.276	0.087
	BOS SPI		-2.598				
	BOS PI-S		0.288				
	BOS P-S		0.505[M]				
Floor Dust Pb Conc. Round 3[2]	ALL GROUPS	1.063[S1]	0.677			0.070	1.867**
	BOS SPI			0.370	1.477[S1]		
	BOS PI-S			1.282[1T]	2.182[S3]		
	BOS P-S			1.668[S1]	2.333[S2]		
Blood Pb Round 1[3]		0.589	0.589	0.589	0.589	0-.589	0.589
		RESPONSE VARIABLE: BLOOD LEAD ROUND 1					
Intercept[1]		10.08[S4]	10.21[S4]	10.99[S4]	8.44[S4]	10.77[S4]	8.26[S4]
Soil Pb Round 1[2]		-0.005	0.029	-0.062	0.394	-0.049	0.400
Dust Pb Round 1[2]		0.392[M]	0.329[M]	0.197	0.629[1T]	0.229	0.693[S3]
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1					
Intercept[4]		2353[S4]	2100[S4]	1689[S3]	1827[S3]	1694[S3]	1875[S3]
Soil Pb Round 1[5]		-0.277[1T]	-0.238[M]	-0.180[M]	-0.203[M]	-0.178	-0.206[M]
Window Dust Pb Round 1[5]		0.0511[S2]	0.0547[S3]	0.0647[S4]	0.0566[S3]	0.0629[S4]	0.0541[S3]
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 3					
Intercept[4]	ALL GROUPS	1103[S4]	1111[S4]	1164[S4]		1107[S4]	
	BOS SPI				345		299
	BOS PI-S				1557		1534
	BOS P-S				959		1004

TABLE 5-36 (cont'd). LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: REGRESSION COEFFICIENTS USING FIXED BLOOD LEAD PERSISTENCE FACTOR FOR FEMALES

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30	UNITS
Soil Pb Conc. Round 3[5]	ALL GROUPS	0.179[S2]	0.168[S1]	0.146[S1]		0.162[S1]		
	BOS SPI				3.636[M]		4.003[M]	
	BOS PI-S				0.064		0.077	
	BOS P-S				0.075		0.065	
Window Dust Pb Conc. Round 3[5]		0.0010	0.0013	0.0020	0.0073	0.0022	0.0065	

[1]Units are μg/dL Pb in blood.
[2]Units are μg/dL Pb in blood per 1000 μg/g Pb in dust or soil.
[3]Units are μg/dL Pb in blood at Round 3 per μg/dL Pb in blood at Round 1.
[4]Units are μg/g Pb in dust.
[5]Units are μg/g Pb in floor dust per μg/g Pb in soil or window dust.

TABLE 5-37. LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: MODEL ASSESSMENT STATISTICS USING FIXED BLOOD LEAD PERSISTENCE FACTOR FOR AGES 18-41 MONTHS

Response Variable	Statistic	MODEL ASSESSMENT STATISTICS MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30	UNITS
Log Blood Pb Round 3	RMSE[1]	0.29243	0.30647	0.32785	0.40901	0.31414	0.34253	
Log Blood Pb Round 1	RMSE	0.33790	0.33689	0.33729	0.34131	0.33631	0.33827	
Log Dust Pb Conc. Round 3	RMSE	0.62465	0.62430	0.62544	0.63384	0.62604	0.63126	
Log Dust Pb Conc. Round 1	RMSE	0.80410	0.80655	0.80448	0.80124	0.80346	0.80124	
All	N*OBJ	20.86	21.07	18.73	16.98	18.74	17.22	

[1]RMSE = Root mean squared error.

TABLE 5-38. LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: REGRESSION COEFFICIENTS USING FIXED BLOOD LEAD PERSISTENCE FACTOR FOR AGES 18-41 MONTHS

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30	UNITS
RESPONSE VARIABLE: BLOOD LEAD ROUND 3								
Intercept[1]	ALL GROUPS	2.01[S2]	1.76[S1]	1.44[1T]	0.51			
	BOS SPI					0.47	-0.38	
	BOS PI-S					2.68[S2]	2.92[S1]	
	BOS P-S					3.02[S2]	3.13[S1]	
Soil Pb Round 3[2]	ALL GROUPS	0.261		-0.574	-1.337[S1]	-0.592	-1.003[1T]	
	BOS SPI		1.597					
	BOS PI-S		0.337					
	BOS P-S		0.183					
Floor Dust Pb Conc. Round 3[2]	ALL GROUPS	0.772	0.850			2.027[1T]	2.800[S1]	
	BOS SPI			1.020	1.903[M]			
	BOS PI-S			2.826[S1]	5.378[S2]			
	BOS P-S			2.975[S1]	5.435[S2]			
Blood Pb Round 1[3]		0.589	0.589	0.589	0.589	0.589	0.589	
RESPONSE VARIABLE: BLOOD LEAD ROUND 1								
Intercept[1]		12.42[S4]	12.18[S4]	12.21[S4]	12.90[S4]	12.00[S4]	12.55[S4]	
Soil Pb Round 1[2]		0.005	-0.047	0.056	-0.105	0.061	-0.052	
Dust Pb Round 1[2]		-0.068	0.013	-0.064	-0.131	-0.018	-0.068	
RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1								
Intercept[4]		1834[S4]	1910[S4]	1520[S4]	1446[S4]	1536[S4]	1522[S4]	
Soil Pb Round 1[5]		-0.152	-0.178[M]	-0.094	-0.027	-0.087	-0.042	
Window Dust Pb Round 1[5]		0.0543[S4]	0.0553[S4]	0.0556[S4]	0.0472[S4]	0.0531[S4]	0.0462[S4]	
RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 3								
Intercept[4]	ALL GROUPS	593[S4]	581[S4]	628[S4]		615[S4]		
	BOS SPI				580[S4]		589[S4]	
	BOS PI-S				684		708	
	BOS P-S				236		206	

TABLE 5-38 (cont'd). LONGITUDINAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: REGRESSION COEFFICIENTS USING FIXED BLOOD LEAD PERSISTENCE FACTOR FOR AGES 18-41 MONTHS

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30	UNITS
Soil Pb Conc. Round 3[5]	ALL GROUPS	0.250[S4]	0.250[S4]	0.244[S4]		0.251[S4]		
	BOS SPI				0.626[S2]		0.533[S1]	
	BOS PI-S				0.144		0.143	
	BOS P-S				0.354		0.406	
Window Dust Pb Conc. Round 3[5]		0.0080[S2]	0.0082[S2]	0.0069[S1]	0.0082[S3]	0.0067[S1]	0.0078[S2]	

[1]Units are μg/dL Pb in blood.
[2]Units are μg/dL Pb in blood per 1000 μg/g Pb in dust or soil.
[3]Units are μg/dL Pb in blood at Round 3 per μg/dL Pb in blood at Round 1.
[4]Units are μg/g Pb in dust.
[5]Units are μg/g Pb in floor dust per μg/g Pb in soil or window dust.

$$(B_{gl}X_{gl} - B_{gr}X_{gr}) - (B_{hl}X_{hl} - B_{hr}X_{hr}) \qquad (5\text{-}23)$$

Model 1 has a single common coefficient for all groups for each pathway coefficient or intercept in the basic model configuration. Model 2 has the same configuration, but instead of a common coefficient for Round 3 blood lead regression on Round 3 soil lead, there are different coefficients in each of the three treatment groups. Otherwise, all parameters are the same in both models. However, when the parameters of the models are estimated from the data, the estimates of the parameters may be different in different models, since parameters cannot be estimated independently of the other parameters. Likewise, Model 10 differs from Model 1 in that the Round 3 blood lead vs dust lead coefficients may be different in different groups. In Model 17, all of the pathway regression coefficients are common among treatment groups, but the effects of the interventions are characterized by different intercepts for blood lead in each treatment group Round 3. Common parameter values in Tables 5-30, 5-32, 5-34, 5-36, and 5-38 are entered as "all" in the parameter cell, whereas the "all" cell is left empty and the treatment group cells are filled in in the models in which separate group parameters are estimated.

The two-equation blood lead and dust lead model for Round 1 was also used for Round 3, and there was an additional component for persistence of a fraction of Round 1 blood lead extending to Round 3. Abatement effects at Round 3 were modelled by separating a single pathway regression coefficient or intercept (group mean) into three separate coefficients, one for each of the treatment groups BOS SPI, BOS PI-S, and BOS P-S. The effect of abatement could then be assessed as a difference among these separate coefficients. All possible combinations of abatement effects could be modelled by one of 32 possible models. Detailed analyses found that only five or at most six of the models gave good results, as assessed by small root mean squared errors (RMSE) for log blood lead and log dust lead in both Round 1 and Round 3. These are shown in Table 5-29. Models with a larger number of free (estimatable) parameters gave smaller overall objective functions across all four state variables. However, assessment of RMSE for the four variables found small differences, at most.

Model 1 did well in all of the analyses. This is the simplest model, assuming that there were no differences in any pathway coefficients or intercepts in any treatment group, and therefore any differences in blood lead or dust lead by Round 3 can only be attributed to differences in soil lead and window dust lead concentrations. In Model 2, this hypothesis is extended to include different regression coefficients in BOS SPI, BOS PI-S, and BOS P-S. However, the Round 3 blood lead versus soil lead regression coefficients shown in Table 5-29 for these groups are 0.517, 0.680, and 0.817 μg/dL per 1000 μg/g lead in soil respectively, compared to 0.719 in Model 1 for all groups combined, none of which are statistically significant or different from each other. Furthermore, as shown in Table 5-29, the overall assessment statistic N * objective is 41.86 in Model 2, only slightly smaller than 42.75 in Model 1, and the RMSE are only slight smaller for Round 3 log blood lead (0.3005 in Model 2 versus 0.3009 in Model 1), for Round 1 log blood lead (0.3651 for Model 2 versus 0.3742 for Model 1), for Round 3 log dust lead (0.6208 in Model 2 versus 0.6215 in Model 1), and for Round 1 log dust lead (0.7909 for Model 2 versus 0.7937 for Model 1).

Models 10 and 11, which allow for different regression coefficients for Round 3 blood lead versus dust lead regression coefficients, showed the best fit for at least three of the four state variables in Table 5-29. RMSE for Round 3 log blood lead was 0.2980 in Models 10 and 11, compared to 0.3009 for Model 1. RMSE for Round 1 log blood lead was 0.3328

for Models 10 and 11, compared to 0.3742 for Model 1. MSE for Round 3 log dust lead was 0.6080 for Model 10, somewhat better than 0.6215 for Model 1, and RMSE for Model 11 was 0.6300 which was somewhat worse than Model 1. RMSE for Round 1 log dust lead was 0.7851 for both Models 10 and 11, lower than 0.7937 for Model 1. In Table 5-30, the Round 3 blood lead versus dust lead regression coefficients for Models 10 and 11 were 0.18 μg/dL per 1000 μg/g dust lead for BOS SPI, 1.27 μg/dL per 1000 μg/g for BOS PI-S, and 1.34 μg/dL per 1000 μg/g for BOS P-S, which were not statistically significant and were not significantly different from each other nor from Model 1. In Model 11, the Round 3 dust lead versus soil lead regression coefficients were allowed to vary by treatment group also. As shown in Table 5-30, the Round 3 dust lead versus soil lead coefficients were -0.06 μg/g dust lead per μg/g soil lead in BOS SPI, 0.30 in Group BOS PI-S, and 0.47, none significant and none significantly different, even though the single dust lead versus soil lead coefficient of 0.20 in Models 1, 2, and 10 was highly significant ($P < 0.0001$ in Models 1 and 2, < 0.002 in Model 10).

Model 17 was the only model providing a good fit in which there were separate intercepts for each group. As shown in Table 5-29, Model 5-17 has a slightly lower RMSE for Round 3 log blood lead than other models, a slightly higher RMSE for Round 1 log blood lead than Model 1, RMSE for Round 3 log dust lead that is somewhat higher than Models 10 and 11 but lower than Models 1 and 2, and slightly higher RMSE for Round 1 log dust lead than Models 10 and 11 but lower than Models 1 and 2. The intercept terms in Table 5-30 are also not significant, but provide easily calculated components of effect size, 4.41 - 1.95 = 2.46 μg/dL for BOS SPI-S versus BOS P-S, 3.28 - 1.95 = 1.33 μg/dL for BOS SPI versus BOS PI-S, 4.41 - 3.28 = 1.13 μg/dL for BOS PI-S versus BOS P-S, which is very similar to the repeated measures ANCOVA effect size estimates in Table 5-5.

The single most sensitive parameter estimate in these models is Round 3 versus Round 1 blood lead regression coefficient, which is extremely consistent at 0.59 to 0.61 μg/dL in Round 3 per μg/dL in Round 1, in all 5 models in Table 5-30. However, this coefficient is statistically significant only in Model 1. Since the persistent effect of pre-abatement blood lead is the largest single component of post-abatement blood lead for most of the children in the sample, we decided to also evaluate models in which this regression coefficient was held fixed at the Model 10 and 11 optimal value 0.589.

Longitudinal SEM with Fixed Estimates of Blood Lead Persistence

Results with the Round 3 versus Round 1 blood lead regression coefficient fixed at 0.589 are shown in Tables 5-31 and 5-32. Table 5-31 shows that fixing the value of this coefficient tends to flatten the effects of separating the other regression model parameters, so that Model 1 provides a smaller RMSE for Round 3 log blood lead than other good models, and smaller or nearly smaller values of RMSE for the other three variables. Model 30 also provides a good fit to all variables, with statistically significant treatment group effects for the blood lead intercept and for the dust lead versus soil lead intercept.

Table 5-32 shows that there are statistically significant post-abatement relationships between blood lead concentration and dust lead concentration in Models 10, 11, 17, and 30. Furthermore, the differences among treatment groups in blood lead versus dust lead regression coefficients in Model 11 is nearly significant, and is marginally significant in Model 10. These differences did not exist for Round 1 blood lead. The effects of abatement are seen in Model 11 in Table 5-32. In the soil abatement group BOS SPI, the typical Round 3 dust lead concentration has been reduced to about 800 μg/g, and there is only a small relationship between dust lead and soil lead (0.19 μg/g dust lead per μg/g soil lead), and in any case the Round 3 soil lead is low in BOS SPI. In the dust abatement group BOS PI-S, the Round 3 dust lead intercept is lower than for BOS SPI or BOS P-S, about 100 μg/g, but the relationship to soil lead is much stronger than in BOS SPI or BOS P-S, 0.39 μg/g dust lead per μg/g soil lead. The difference between BOS SPI and BOS PI-S is that the soil lead is much lower in BOS SPI, so that dust lead tends to be lower in BOS PI-S than in BOS SPI. On the other hand, while the Round 3 dust lead intercept and the Round 3 dust lead versus soil lead regression coefficient are similar in BOS SPI and in BOS P-S, Round 3 soil lead is much lower in BOS SPI, as is Round 3 dust lead. Therefore, the soil lead abatement effect appears to be related simply to reduction of lead concentrations in soil and dust. The partial effect of the dust lead abatement in BOS PI-S may have a component that could be attributed to a change (possibly temporary) in the soil lead to dust lead pathway.

A simple calculation of effect size based on separate group lead intercept terms for Round 3 blood lead is also informative. In Table 5-32, for Model 17, the BOS SPI versus BOS P-S effect is 2.10 - 0.24 = 1.86 μg/dL (nearly significant), the BOS SPI versus

BOS PI-S effect is 1.56 μg/dL, and the BOS PI-S versus BOS P-S effect is not significant. In Model 30, the BOS SPI versus BOS P-S effect is significant, 2.53 μg/dL, and the BOS PI-S versus BOS P-S effect of 2.54 μg/dL is nearly significant. There is a significant relationship between blood lead and dust lead in these two models, and significant (possibly different) relationships between dust lead and soil lead, so that there is again evidence of the operation of soil lead abatement by a soil lead to dust lead to blood lead pathway in the Boston study.

Effects of Gender in Longitudinal SEM

Sensitivity of the LSEM models was also evaluated by stratifying the sample by gender and fitting different models for males and females. Results for males are shown in Tables 5-33 and 5-34 and results for females in Tables 5-34 and 5-35. As shown in Table 5-33, Models 11 and 30 provided a good fit to blood lead data for males in Rounds 1 and 3. Table 5-35 shows a somewhat different pattern for females, with Models 1 and 10 providing a somewhat better fit to blood lead than models 11 and 30.

Table 5-34 shows that the Round 3 blood lead versus dust lead regression coefficients for males are less significant, and the Round 3 blood lead versus soil regression coefficients much more significant in Models 1 and 2 in the male subgroup than in the whole sample. Table 5-36 presents a different finding for females, with blood lead versus soil lead coefficients marginally significant or not significant in most models, and blood lead versus dust lead coefficients significant or highly significant in most models. This suggests that direct soil lead exposure may be more important for boys and dust lead exposure inside the home somewhat more important for girls in the Boston study.

Effect size estimates for males may be taken from Model 30 in Table 5-34. The effect of BOS SPI versus BOS P-S is 1.14 μg/dL (not significant) and the effects of BOS SPI versus BOS PI-S is 1.61 μg/dL (marginally significant). The effect of BOS PI-S versus BOS P-S is not statistically significant. However, this must be combined with an assessment of differential abatement effects in Table 5-34 on the dust lead versus soil lead relationship in the male residences, where a very large and statistically significant Round 3 relationship between dust lead and soil lead exists in the control group, 0.93 μg/g lead in dust per μg/g lead in soil, but not in the two abatement groups BOS PI-S and BOS SPI.

Effect size estimates for females may be taken from Model 17 in Table 5-36. The effect of BOS SPI versus BOS P-S is 2.13 μg/dL (significant) and the effects of BOS SPI versus BOS PI-S is 0.91 μg/dL (not significant). The effect of BOS PI-S versus BOS P-S is not statistically significant. In Table 5-36, the dust lead versus soil lead relationship in the female residences is statistically significant at Round 3, 0.162 μg/g dust lead per μg/g soil lead, whereas in male residences in Table 5-34, the Model 17 relationship between dust lead and soil lead is larger and much more significant, 0.25 μg/g lead in dust per μg/g lead in soil, but not in the two abatement groups BOS PI-S and BOS SPI. However, the differential treatment group relationships in Models 11 or 30 are strikingly different between males and females. The BOS SPI Round 3 dust lead versus soil lead coefficient for females is very large, about 4 μg/g dust lead per μg/g soil lead, and the coefficients are negligible for BOS PI-S and BOS P-S, whereas the estimated BOS SPI coefficient for males is small in BOS SPI, about 0.2, and large for BOS P-S, about 0.9. These differences may be statistical artifacts, since homes were abated similarly whatever the gender of the resident children. An alternative hypothesis, that the dust lead versus soil lead relationship depends on the gender of the child residing in the house, seems implausible. Additional studies of gender effects may be of considerable scientific interest. However, it is clear that soil lead abatement is associated with reduced childhood blood lead in the Boston study in both male and female children, even if there is some possibility that the soil and dust exposure processes may differ by gender.

Longitudinal SEM by Age Group

There were not enough children for separate analyses of Boston children in age groups < 18 months or > 41 months. The results for 18-41 month old children shown in Tables 5-37 and 5-38 were very similar to those for the group as a whole, with Models 1, 2, and 17 providing the best fit to blood lead data. Effect size estimates for Model 17 were also similar to repeated measures ANOVA results, 2.55 μg/dL for BOS SPI versus BOS P-S and 2.21 μg/dL for BOS SPI versus BOS PI-S (both statistically significant). There appears to be a significant group difference in the relationship between dust lead and soil lead at Round 3, with a significantly stronger relationship in the BOS SPI group and a similar but weaker relationship in the BOS P-S group compared to the BOS PI-S group.

5.6.2 Cincinnati Study Longitudinal Structural Equation Models

The very simple model that assumes the same relationships among blood lead, dust lead, and soil lead in all neighborhoods (called Model 1) provided a reasonably adequate description of the variability in the data. A large number of alternative models were investigated, but only Models 2, 5, and 6 significantly improved the goodness of fit. The most important of these was Model 5, which tested the hypothesis that there were differences in average residential floor dust lead among the five neighborhoods at Round 4. The question of whether these differences should be attributed to the soil or dust lead abatements is discussed below. The models with neighborhood group mean differences in blood lead that were analogous to these modifications were used to calculate effect sizes (Models 2 and 6). Other modifications that somewhat improved the fit were that there were neighborhood differences in neighborhood mean floor dust lead concentrations at Round 1 that could not be attributed to a common relationship of floor dust lead to soil lead (called Models J1 to J6 respectively).

The most sensitive parameter in the model was regression coefficient of Round 4 blood lead on Round 1 blood lead, which we interpreted earlier as the blood lead persistence parameter. The optimal value of the parameter for fitting all four state variables (log blood lead at Rounds 1 and 4, log dust lead at rounds 1 and 4) usually provided a somewhat inferior fit for Round 4 blood lead, so we modified the fitting procedure first to estimate the value of this coefficient that optimized prediction of Round 4 blood lead, then optimized all of the other parameters. The results are shown in Table 5-39 and 5-40. Table 5-39 shows the model assessment statistics that were used, including the global objective function for the iterated generalized method of moments procedure and the RMSE of the four state variables that were fitted. The regression models for blood lead at Rounds 1 and 4, and the dust lead regression models are shown in Table 5-40.

The optimized models all suggest values of the blood lead persistence parameter that are very similar to that in the Boston longitudinal structural equation model, in the range 0.54 to 0.63. The models also show that adjustments for neighborhood differences in blood lead and floor dust lead at each round clarify the pattern of effects. In Table 5-40, it is clear that blood lead intercept terms differ substantially across the neighborhoods even after

TABLE 5-39. LONGITUDINAL STRUCTURAL EQUATION MODELS FOR CINCINNATI STUDY: MODEL ASSESSMENT STATISTICS USING FIXED BLOOD LEAD PERSISTENCE FACTOR

		MODEL ASSESSMENT STATISTICS						
Response Variable	Statistic	MODEL 1	MODEL 5	MODEL 6	MODEL J5	MODEL J6	MODEL 30	UNITS
Log Blood Pb Round 3	RMSE[1]	0.50410	0.51099	0.53409	0.51589	0.49415		
Log Blood Pb Round 1	RMSE	0.52740	0.52134	0.53216	0.76285	0.78434		
Log Dust Pb Conc. Round 3	RMSE	0.88351	0.84617	0.83267	0.83566	0.82889		
Log Dust Pb Conc. Round 1	RMSE	0.72687	0.73471	0.73004	0.82747	0.84609		
All	N*OBJ	32.59	22.22	17.82	20.16	19.33		

[1]RMSE = Root mean squared error.

adjustment for individual household dust lead concentrations. The Round 4 intercepts are large and statistically significant for CIN NT(M) and CIN SEI(P) in Models 2, J2, and J6, and large for CIN NT(M) in Model 6. At least some of the children in CIN NT(M) (and possibly CIN SEI(P)) may have been exposed to a lead source or medium contaminated with lead, other than soil and floor dust, to which most children in the other neighborhoods were not exposed. The adjustment for changes in dust lead are suggested in Table 5-40, which suggests large increases in average dust lead in CIN I-SE(D), CIN I-SE(F), and CIN SEI(P) from Round 1 to Round 4, everything else being equal.

5.6.3 Calculating Effect Sizes from Longitudinal Structural Equation Models

This section illustrates how effects sizes can be calculated from the results for certain longitudinal structural equation models for Boston and Cincinnati. The effect size comparisons for Boston are shown in Table 5-41. The Boston results are based on Table 5-32. While several different models were evaluated, Model 17 offered the smallest global objective function and among the smallest RMSE of all models fitted by the Iterated

TABLE 5-40. LONGITUDINAL STRUCTURAL EQUATION MODELS FOR CINCINNATI STUDY: REGRESSION COEFFICIENTS USING FIXED BLOOD LEAD PERSISTENCE FACTOR

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 5	MODEL 6	MODEL J5	MODEL J6	UNITS
		RESPONSE VARIABLE: BLOOD LEAD ROUND 4					
Intercept[1]	ALL GROUPS	1.73[S2]	1.16[1T]		0.46		
	CIN I-SE(D)			-0.14		1.19	
	CIN I-SE(F)			-1.50[M]		0.66	
	CIN NT(G)			-1.00[S1]		0.49	
	CIN NT(M)			3.27[S1]		3.27[S1]	
	CIN SEI(P)			-0.09		1.84[1T]	
Floor Dust Pb Round 4[2]		3.84[S2]	4.70[S2]	5.70[S4]	4.74[S3]	3.34[S3]	
Soil Pb Round 4[2]		-0.05	0.67		0.90		
Blood Pb Round 1 (Fixed)[3]		0.4824	0.5221	0.6953	0.5456	0.5801	
		RESPONSE VARIABLE: BLOOD LEAD ROUND 1					
Intercept[4]	ALL GROUPS	8.84[S4]	7.35[S4]	6.73[S4]			
	CIN I-SE(D)				10.25[S4]	10.61[S4]	
	CIN I-SE(F)				8.98[S4]	9.34[S4]	
	CIN NT(G)				7.02[S4]	7.05[S4]	
	CIN NT(M)				6.82[S4]	6.42[S4]	
	CIN SEI(P)				6.87[S4]	7.23[S4]	
Floor Dust Pb Round 1[2]		3.00[S1]	2.49[1T]	4.02[S1]	4.52[S3]	3.84[S2]	
Soil Pb Round 1[2]		-0.39	3.04[S1]	2.68[1T]			
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1					
Intercept[4]	ALL GROUPS	150[S4]	156[S4]	136[S4]			
	CIN I-SE(D)				166[S4]	150[S3]	
	CIN I-SE(F)				74[1T]	51[M]	
	CIN NT(G)				115[S1]	108[S1]	
	CIN NT(M)				217[S4]	170[S4]	
	CIN SEI(P)				132[S3]	128[S2]	
Window Dust Pb Round 1[5]		0.0292[S3]	0.0255[S3]	0.0324[S3]	0.1153[S4]	0.1293[S4]	
Soil Pb Round 1[5]		0.188[S2]	0.157[S2]	0.166[S2]			

TABLE 5-40 (cont'd). LONGITUDINAL STRUCTURAL EQUATION MODELS FOR CINCINNATI STUDY: REGRESSION COEFFICIENTS USING FIXED BLOOD LEAD PERSISTENCE FACTOR

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 5	MODEL 6	MODEL J5	MODEL J6	UNITS
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1					
Intercept[4]	ALL GROUPS	150^{S4}	156^{S4}	136^{S4}			
	CIN I-SE(D)				166^{S4}	150^{S3}	
	CIN I-SE(F)				74^{1T}	51^{M}	
	CIN NT(G)				115^{S1}	108^{S1}	
	CIN NT(M)				217^{S4}	170^{S4}	
	CIN SEI(P)				132^{S3}	128^{S2}	
Window Dust Pb Round 1[5]		0.0292^{S3}	0.0255^{S3}	0.0324^{S3}	0.1153^{S4}	0.1293^{S4}	
Soil Pb Round 1[5]		0.188^{S2}	0.157^{S2}	0.166^{S2}			
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 4					
Intercept[4]	ALL GROUPS	324^{S4}					
	CIN I-SE(D)		235^{S4}	235^{S4}	295^{S4}	293^{S4}	
	CIN I-SE(F)		227^{S4}	284^{S3}	212^{S4}	210^{S3}	
	CIN NT(G)		79^{S3}	82^{S4}	86^{S4}	78^{S4}	
	CIN NT(M)		327^{1T}	53	89	71	
	CIN SEI(P)		254^{S4}	235^{S4}	295^{S4}	237^{S4}	
Window Dust Pb Round 1[5]		0.0826^{S4}	0.0813^{S4}	0.0781^{S4}	0.0740^{S4}	0.0832^{S4}	
Soil Pb Round 1[5]		-0.121^{S1}					

[1]Units are μg/dL Pb in blood.
[2]Units are μg/dL Pb in blood per 1000 μg/g Pb in dust or soil.
[3]Units are μg/dL Pb in blood at Round 3 per μg/dL Pb in blood at Round 1.
[4]Units are μg/g Pb in dust.
[5]Units are μg/g Pb in floor dust per μg/g Pb in soil or window dust.

Generalized Method of Moments (ITGMM) method in SAS PROC MODEL. In Model 17, the three treatment groups are assumed equal at Round 1. However, at Round 3, the three groups are assumed to have different blood lead intercept values due to differences in treatment. If G_1 denotes the overall blood lead intercept at Round 1and G_{g3} denotes the

TABLE 5-41. COMPARISON OF STATISTICAL METHODS FOR BOSTON STUDY: REDUCTION IN BLOOD LEAD (E_r) BETWEEN ROUNDS 1 AND 3

Study Group		Statistical Method	
Abate Versus Control		Repeated Measures Analysis of Variance[1]	Longitudinal Structural Equation Model[1]
BOS SPI	BOS P-S	1.87[S2]	1.86
BOS PI-S	BOS P-S	0.33	0.30
BOS SPI	BOS PI-S	1.54[S1]	1.56

[1]Units are μg/dL Pb in blood.

intercept for group g at Round 3, then the general form of the two blood equations for Model 17 as fitted are:

$$Blood_{i1} = G_1 + B_1 * Dust_{i1} + F_1 * Soil_{i1} \qquad \text{for all groups}$$

$$Blood_{i3} = G_{g3} + Blood_{i1} * A_{13} + B_3 * Dust_{i3} + F_3 * Soil_{i3} \qquad \text{In group g,}$$

where

$Blood_{i1}$, $Blood_{i3}$ = blood lead concentration in rounds 1 and 3 respectively,

$Dust_{i1}$, $Dust_{i3}$ = floor dust lead concentration in rounds 1 and 3,

$Soil_{i1}$, $Soil_{i3}$ = soil lead concentrations in rounds 1 and 3,

B_1, B_3 = regression coefficient of blood lead on floor dust lead at rounds 1 and 3,

F_1, F_3 = regressions coefficients of blood lead on soil lead at rounds 1 and 3,

A_{13} = regression coefficient of blood lead at round 3 on blood lead at round 1.

The "base" effect size estimates can be calculated as the difference of changes in intercepts.

$$E_{[SPI,\ P\text{-}S]} = (G_1\ (1\text{-}A_{13}) - G_{[SPI.3]}) - (G_1(1\text{-}A_{13}) - G_{[P\text{-}S.3]})$$
$$= G_{[P\text{-}S.3]} - G_{[SPI.3]}) = 2.10 - 0.24 = 1.86\ \mu g/dL.$$

where the adjustment for removable blood lead in Phase 1 is $(1 - A_{13})$. The other LSEM entries in Table 5-41 are similarly calculated from Model 17 Round 3 blood lead intercept terms in Table 5-31. Additional adjustments of effect size can be made using dust lead and soil lead regression coefficients to standardize the comparisons.

The Cincinnati analyses are based on Model J6 in Table 5-39 and are more complicated because the neighborhoods are initially quite different with respect to both soil lead and dust lead concentrations. In general, the ITGMM objective function is greatly reduced by including separate intercept terms at both Round 1 and Round 4, both for blood lead and dust lead. Thus the model fitted to the blood lead data are in the form:

$$Blood_{i1} = G_{g1} + B_1 * Dust_{i1}$$

$$Blood_{i4} = G_{g4} + Blood_{i1} * A_{14} + B_4 * Dust_{i4} \quad \text{in group g,}$$

so that the effect sizes for the "base" model are in the form

$$E_{[CIN\ NT(G),\ CIN\ NT(M)]} = \{G_{[CIN\ NT(G),1]}\ (1\text{-}A_{14}) - G_{[CIN\ NT(G),4]}\} - \{G_{[CIN\ NT(M),1]}\ (1\text{-}A_{14}) - G_{[CIN\ NT(M),4]}\}$$
$$= \{\ 7.05\ (1 - 0.5801) - 0.49\} - \{6.42\ (1 - 0.5801) - 3.27\}$$
$$= 2.47 - (-0.57) = 3.04\ \mu g/dL.$$

The entries in Table 5-43 are the group intercept changes adjusted for removable blood lead. The final effect sizes in Table 5-42 were calculated in the same way as differences of changes in Table 5-43.

The right columns of Table 5-43 shows the effect of adding a dust lead adjustment. The blood lead effect size adjustments were calculated from the blood lead - dust lead regression coefficients B1 and B4, and the median or average dust lead concentrations in each neighborhood at Rounds 1 and 4 respectively. The defining equation is:

TABLE 5-42. COMPARISON OF STATISTICAL METHODS FOR CINCINNATI STUDY: REDUCTION IN BLOOD LEAD (E_r) BETWEEN ROUNDS 1 AND 4

STUDY GROUP		STATISTICAL METHOD		
			LONGITUDINAL STRUCTURAL EQUATION MODEL[1]	
ABATE VS CONTROL		REPEATED MEASURES ANALYSIS OF VARIANCE[1]	BASE MODEL	ADJUSTED FOR MEDIAN DUST
CIN NT(G)	CIN NT(M)	3.58	3.04	3.67
CIN SEI (P)	CIN NT(G)	-2.56	-1.27	-1.45
CIN SEI (P)	CIN NT(M)	1.02	1.77	2.22
CIN SEI (P)	CIN I-SE(D)	-2.43	-2.07	-1.71
CIN SEI (P)	CIN I-SE(F)	-1.20	-2.06	-1.64

[1]Units are μg/dL Pb in blood.

$$ED_{[CIN\ NT(G),CIN\ NT(M)]} = \{B_1*(Dust_{i1}\text{-CIN NT(G)}) - B_4*(Dust_{i4}\text{-CIN NT(G)})\} - \{B_1*(Dust_{i1}\text{-CIN NT(M)}) - B_4*(Dust_{i4}\text{-CIN NT(M)})\}.$$

The entries in Table 5-43 are the estimated changes in blood lead attributable to dust lead between Round 1 and Round 4. There is no "standard" standardization for dust lead or other environmental covariates. Blood lead changes and effects sizes were also calculated using dust lead intercepts from Model 17 in Table 5-39, and were similar to those shown for mean or median dust lead. The effects in Table 5-42 show a large Phase 1 blood lead reduction in CIN NT(G) compared to CIN NT(M), a moderately large blood lead reduction in CIN SEI(P) compared to CIN NT(M), and and moderate to large blood lead increases in CIN SEI(P) compared to CIN I-SE(D), CIN I-SE(F), or CIN NT(G).

TABLE 5-43. CALCULATION OF DUST LEAD ADJUSTMENT TO BLOOD LEAD REDUCTION OF THE BOSTON AND CINCINNATI STUDIES FROM PARAMETERS OF THE STRUCTURAL EQUATION MODEL IN TABLES 5-32 AND 5-40

	Parameter From Table 5-32 (Boston) or 5-40 (Cincinnati)											
	Median Floor Dust Lead		Median Blood Lead Adjustment		Change in Median Blood Lead		Blood Lead Intercept		Removable Blood Lead	Intercept Change RD 1 RD 4		
Study Group	RD1	RDP[1]	RD1	RDP[1]	For Dust	For Soil	RD1	RDP[1]		No Adjust	Adjust For Median Dust	Adjust For Median Soil and Dust
	X_1	X_p	B_1X_1	B_pX_p	Col 3 - Col 4	F_1Z_1 - F_1Z_p	(G_1)	(G_p)	$G_1(1-A_{1p})$	col 9-col 8	col 10 + col 5	col 10 + col 5 + col 6
	Col 1	Col 2	Col 3	Col 4	Col 5	Col 6	Col 7	Col 8	Col 9	Col 10	Col 11	Col 12
BOS SPI	2420	876	-0.12	1.53	-1.65	-0.33	12.66	0.24	5.20	4.96	3.31	2.98
BOS PI-S	2582	1198	-0.13	2.10	-2.23	0.19	12.66	1.80	5.20	3.40	1.17	1.36
BOS P-S	2536	1504	-0.13	2.64	-2.77	0.22	12.66	2.10	5.20	3.10	0.33	0.55
CIN I-SE(D)	414	498	1.69	1.66	-0.06		10.61	1.19	4.46	3.27	3.21	
CIN I-SE(F)	469	392	1.80	1.31	0.49		9.34	0.66	3.92	3.26	3.75	
CIN NT(G)	187	187	0.72	0.60	0.12		7.05	0.49	2.96	2.47	2.59	
CIN NT(M)	392	392	1.51	1.45	0.06		6.42	3.27	2.70	-0.57	-0.51	
CIN SEI (P)	366	366	1.41	1.60	-0.19		7.23	1.84	3.04	1.20	1.01	

[1]For Boston, P=Round 3, for Cincinnati, P=Round 4.

Cols 1, 2: Data are from Appendix A, units are μg/g.

Col 3: Units are μg/dL, calculated as (Col 1)(-0.050 μg/dL per 1000 μg/g) for Boston (from Table 5-32, model 17), or (Col 1)(3.84 μg/dL per 1000 μg/g) for Cincinnati (Table 5-40, model J6).

Col 4: Units are μg/dL, calculated as (Col 2)(1.752 μg/dL per 1000 μg/g) for Boston (from Table 5-32, model 17), or (Col 2)(3.34 μg/dL per 1000 μg/g) for Cincinnati (Table 5-40, model J6).

Col 6: Analogous calculation for Boston, -0.150 μg/dL per 1000 μg/g in Round 1 and -0.263 μg/dL per 1000 μg/g in Round 3, median soil lead concentrations 2413 and 113, 2477 and 2148, 2268 and 2115.

5.7 SUMMARY OF RESULTS OF STATISTICAL ANALYSES

5.7.1 Synthesis of Results by Repeated Measures ANOVA and Longitudinal Structural Equations Modeling

The different statistical methods used in this study produce quantitatively similar results, with no essential qualitative differences in conclusions from the studies. The preceeding Tables 5-41 and 5-42 compare effect size estimates from repeated measures ANOVA analyses and longitudinal structural equations models for Boston and Cincinnati. Table 5-41 shows the estimated Phase 1 blood lead reductions of the soil abatement group (BOS SPI) and dust abatement group BOS PI-S vs the Phase 1 control group BOS P-S. The effect sizes are almost identical, even though the estimates from Model 17 are based on differences in intercepts after adjustments implicit in a four-equation model involving floor dust lead and blood lead from Rounds 1 and 3.

Table 5-42 shows the estimated Phase 1 reduction in blood lead of one of the Cincinnati no-treatment groups (CIN NT(G)) vs the other no-treatment neighborhood (CIN NT(M)), and the Phase 1 soil abatement neighborhood (CIN SEI(P)) vs these two neighborhoods and the two Phase 1 neighborhoods that received only interior dust abatement (CIN I-SE(D) and CIN I-SE(F)). The longitudinal SEM effects were calculated from Model J-6 in Table 5-39 The effect size estimates from Model J6 were calculated in two different ways. The "base" model calculated treatment group effects as the difference in the change from Round 1 to Round 4 in blood lead model intercept terms between the two neighborhoods. The model "adjusted for mean dust lead" adds analogous terms using the mean dust lead concentrations in the neighborhoods at each round to adjust the overall difference. The worksheet for this calculation is shown in Table 5-43. The differences between methods are larger than for Boston, but less than 1.3 μg/dL in each group. The largest differences reduce the advantage of CIN NT(G) over CIN SEI(P) from 2.6 μg/dL in repeated measures ANOVA to 1.4 μg/dL in longitudinal SEM, and increased the advantage of CIN SEI(P) over CIN NT(M) from 1.0 μg/dL to 2.2 μg/dL. The difference between control neighborhoods CIN NT(G) and CIN NT(M), the largest statistically significant difference between any two neighborhoods, remained at about 3.6 to 3.7 μg/dL by either method.

The longitudinal structural equation Model 17 for Boston, the parameters for which are shown in Table 5-33, is sketched in Figure 5-48. The model shows intercepts and regression

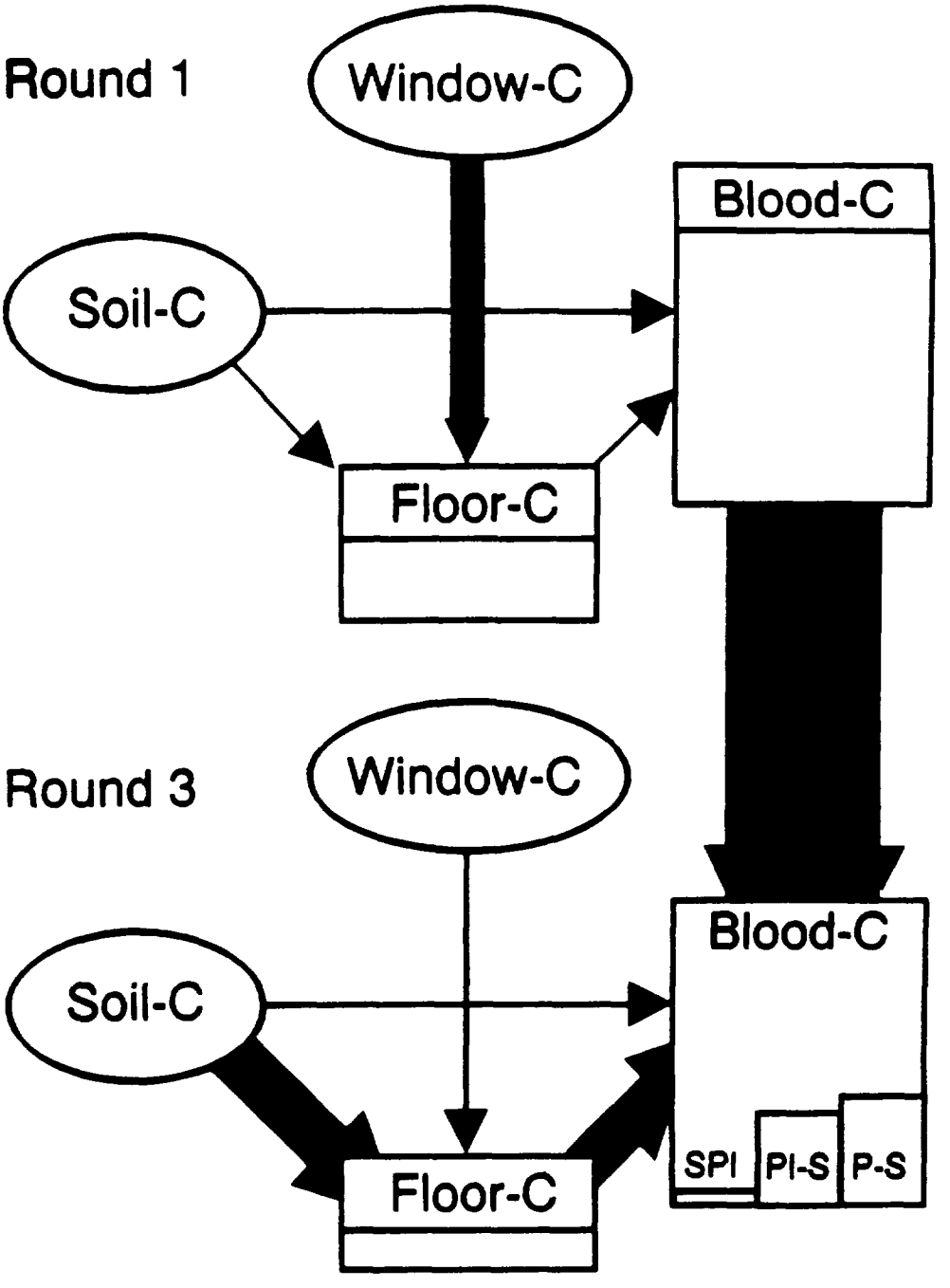

Figure 5-48. Pathway diagram showing the results of the longitudinal structural equation Model 17 for the Boston study, from Table 5-32, using the standard terminology of Figure 5-44. Width of the arrow is proportional to the regression coefficient for Model 17 in Table 5-32. Regression coefficients shown by thin lines are not statistically significant ($P > 0.2$), and those shown by shaded thick lines are statistically significant ($P < 0.05$). Height of the bars in the boxes is proportional to the intercept of the regression model, with separate intercepts for each treatment group shown in the Round 3 blood lead box.

coefficients that have been scaled so as to exhibit the relationships among lead in soil, window dust, floor dust, and blood, before and after Phase 1 abatement. In Figure 5-48, the width of the arrows connecting each of the independent and dependent variables, and the height of the bars in each box is proportional to parameters in Table 5-32. In the pre-abatement model (Round 1), soil lead concentration has little relationship to blood lead or dust lead. This is contrary to experience in other urban areas and may reflect a selection effect. Window dust lead is correlated with interior floor dust lead, however. The post-abatement model (Round 3) shows several strong statistical relationships. The most important predictor of post-abatement blood lead is the pre-abatement blood lead, which includes prior exposures from elevated soil lead and dust lead concentrations, but post-abatement dust lead is also a significant predictor of blood lead. Window lead is also a statistically significant predictor of dust lead, although much smaller in magnitude than soil lead in the post-abatement data.

While post-abatement soil lead has a small and non-significant direct relationship to blood lead, the indirect relationship of soil lead to blood lead through house dust is statistically significant. The combined effect is small, but significant: 0.214 μg dust Pb/g soil and 1.752 μg blood Pb/ 1000 g dust corresponds to a composite effect (0.214 * 1.752) = 0.375 μg/dL per 1000 μg/g in soil. However, this corresponds only to differentials in blood lead associated with differences in soil lead at Round 3. There is also an overall effect characterized by the abatement group intercepts, which were not significantly different at Round 1, but are significantly different at Round 3, as shown by the height of the bars in the intercept box. The control group BOS P-S had the highest post-abatement intercept, 2.10 μg/dL, and the soil abatement group BOS SPI had the lowest intercept, 0.24 μg/dL. The difference of 2.10 - 0.24 = 1.86 μg/dL is the intercept difference effect size. However, in addition to this, there is additional benefit to the group BOS SPI because of the lower dust lead and soil lead concentrations in most of the BOS SPI residences. The median difference is about 2.43 μg/dL, taking differences in median levels of soil lead and dust lead into account.

Figure 5-49 shows Cincinnati LSEM Model 5, the parameters for which are given in Table 5-40. The scaling is the same as in the Boston model, Figure 5-48, except for the floor dust concentrations which are shown 1/10 as high as in the Boston figure. The floor

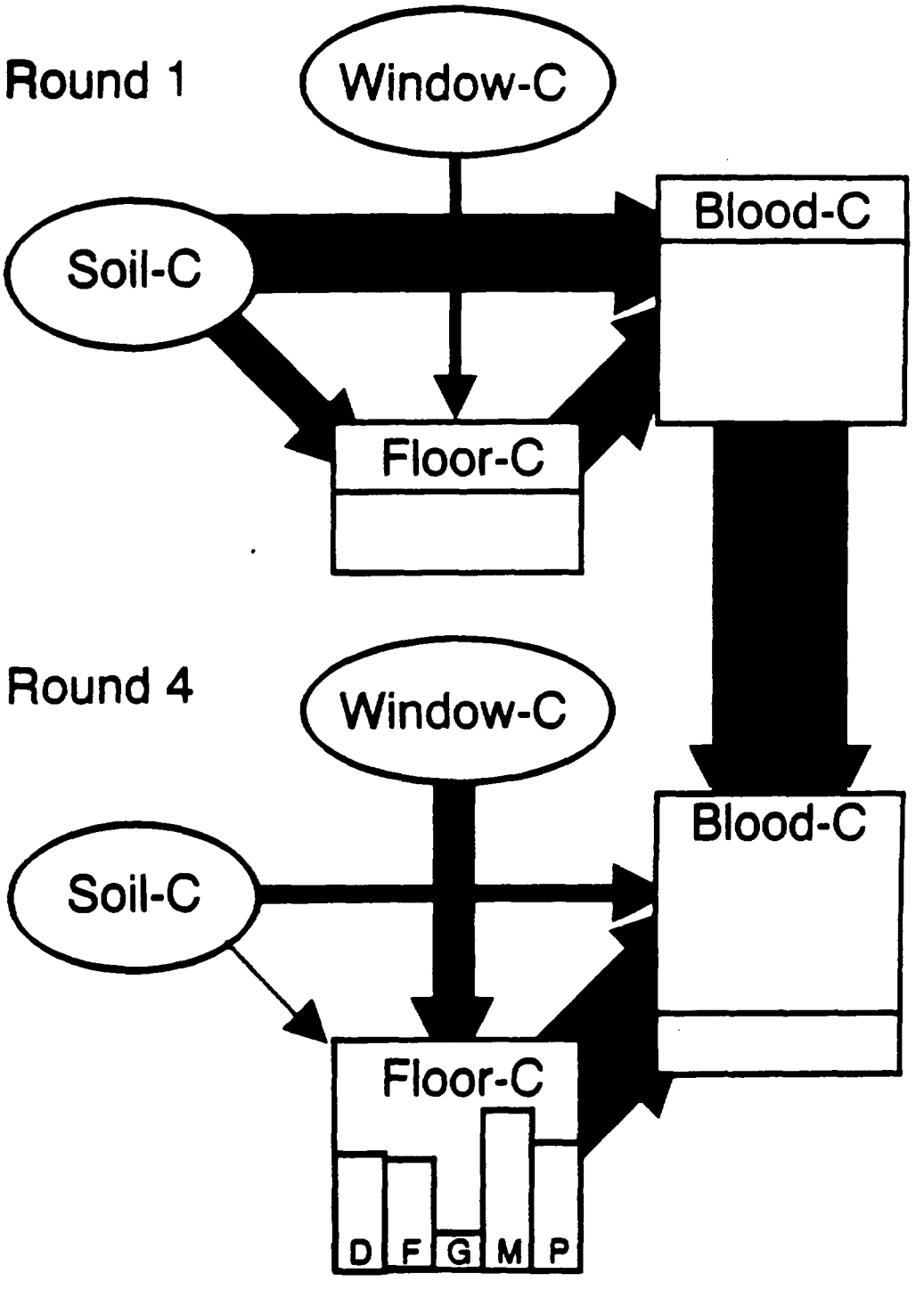

Figure 5-49. Pathway diagram showing the output from the longitudinal structural equation Model 5 for the Cincinnati study, from Table 5-40. Format is the same as Figure 5-48. Width of the arrow is proportional to the regression coefficient for Model 5 in Table 5-39.

dust concentration intercept in the Cincinnati Round 1 is 156 $\mu g/g$, compared to 1520 $\mu g/g$ in the Round 1 Boston model 17. The Cincinnati model 5 differs from the Boston model 17 in that the inclusion of group mean differences for floor dust lead at Round 4 significantly improved the goodness of fit. Floor dust lead differs significantly among groups. However, Model 5 suggests that differences in neighborhood soil lead and household dust lead are adequate for predicting mean post-abatement blood lead concentrations, so the Model 5 figure shows a large and statistically significant effect of household dust lead, taking into account group differences in dust lead. Neighborhood soil lead differences do not add significant predictiveness at postabatement Round 4, once dust lead is taken into account. This is strikingly different than the Round 1 preabatement model for Cincinnati, which shows large and statistically significant relationships from soil lead to dust lead, and large, distinguishable, and statistically significant effects of neighborhood soil lead and household dust lead on blood lead. The dashed line connecting soil lead to floor dust lead at Round 4 shows that a relationship exists, but cannot be estimated well because of the confounding of dust intercepts with neighborhood soil lead. Window lead is a statistically significant predictor of floor dust lead in Round 1, as well as a larger and more significant predictor of dust lead at Round 4.

Figure 5-50 shows a more disaggregated version of Figure 5-49. The parameters are derived from Model J6 in Table 5-40. Since treatment group intercepts are used for floor dust lead and blood lead in Rounds 1 and 4, direct soil lead effects are not estimatable. The relation of floor dust lead to window lead is large and statistically significant, both in Round 1 and Round 4. The relationship of floor dust lead to blood lead is large, statistically significant, and quantitatively rather similar in Rounds 1 and 4, even though dust lead concentrations were typically much lower in Cincinnati than in Boston. The change in blood lead from Round 1 to Round 4 shows little relationship to abatement group, decreasing sharply in control group CIN NT (G) and increasing in control group CIN NT (M), decreasing in dust abatement groups CIN I-SE (D) and CIN I-SE (F), but decreasing less in the soil abatement group SEI (P). The environmental pathways are significant, as one might have expected from studies carried out in Cincinnati a decade earlier (Bornschein et al., 1985). Restricting the study to fully rehabilitated residences probably reduced lead paint effects, but other external changes in lead exposure (manifested in part through window dust

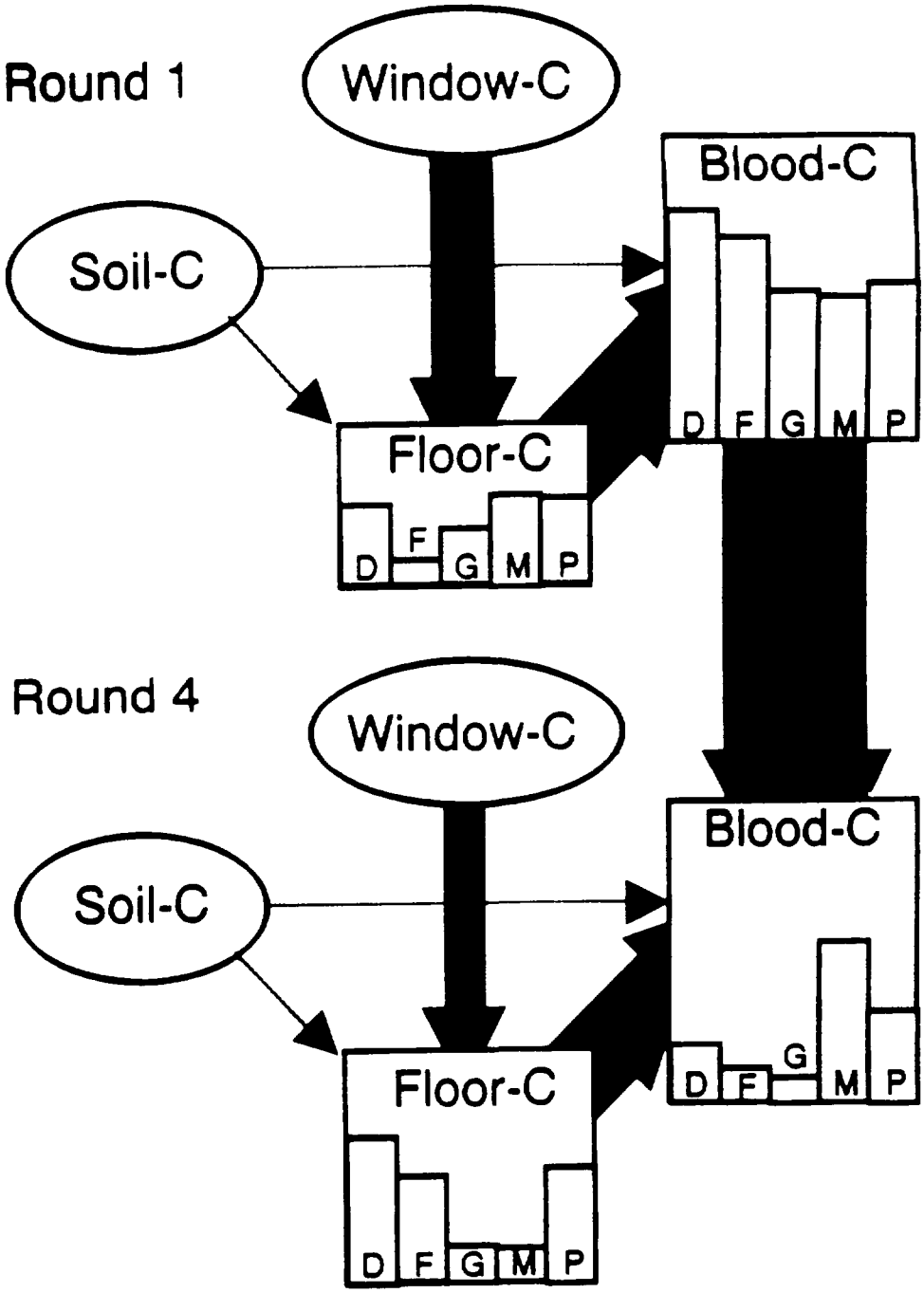

Figure 5-50. Pathway diagram showing the output from longitudinal structural equation Model J6 for the Cincinnati study. Format is the same as Figure 5-48. Pathways shown by dashed lines cannot be estimated using only neighborhood-average soil lead concentration. Height of the bars in the boxes is proportional to the intercept of the regression model, with separate intercepts for each treatment group shown in the Round 1 and 3 floor dust lead and blood lead boxes.

lead concentrations) seemed to be more important factors than soil and exterior dust abatement or Round 4 floor dust lead. Additional analyses to evaluate the role of other external changes would be useful.

5.7.2 Summary of Results by Study

This integrated assessment of the USLADP includes a reevaluation of the results of the analyses carried out by the original investigators and of the conclusions reached by the investigators based on their analyses. While the numerical results of their analyses have been largely confirmed here, other interpretations of the results are also consistent with these numerical findings and, in some cases, may be more plausible than the conclusions published by the investigators. The results of the original investigations have also been extended here by carrying out additional analyses, using a consistent set of powerful analytical techniques not available when the original reports were published.

5.7.2.1 Boston Study

The Boston study shows very clear evidence of an effect of soil lead abatement in reducing blood lead in children currently residing in lead-contaminated housing. The effect was detected in the whole group of children that received soil abatement, amounting to about 1.9 μg/dL or 17 percent on average, but was much larger in the subgroup of children ages 1.5 to 3.5 years, amounting to about 2.5 μg/dL or 20% of their mean starting blood lead concentration. Since these children had high lead burdens to start with, the blood lead reduction was actually about half of the potentially removable blood lead. This is based on our estimate that the contribution of pre-abatement blood lead to post-abatement blood lead was 59% of the pre-abatement blood lead concentration, so that only about 41% of the pre-abatement blood lead was potentially removable. The contribution of pre-abatement body burden occurs by resorption of pre-abatement lead stored in the skeleton back into the blood after the soil lead abatement had reduced environmental lead exposure.

The Boston study also included a treatment group that received only interior dust abatement, whereas the soil abatement group received both soil and dust abatement. The reference group or control group in the Boston study received neither soil abatement nor dust abatement, but all three groups received interior paint stabilization, a potentially non-trivial

intervention. Therefore, even the control group in the Boston study was not entirely a "no treatment" group, and these children may have benefitted as well from the interior paint stabilization. The dust lead abatement group showed a large transient reduction in blood lead at in the early postabatement stage of the study, similar to the blood lead reduction achieved in the soil abatement group, but by the end of Phase 1 of the study, the blood lead reduction in the dust abatement group was only 1.3 μg/dL. The blood lead reduction in the soil abatement group persisted throughout the study, including into Phase 2.

In Phase 2 of the study the roles of the treatment groups were reversed. The original dust abatement and control groups were given soil and interior dust abatement. The first of these two groups, which received two interventions (dust abatement in Phase 1 and soil abatement in Phase 2), showed a further striking reduction of 3.8 μg/dL or about 40% compared to the group that received only Phase 1 soil abatement, and about 2 μg/dL or 20% reduction compared to the group that received no abatement in Phase 1 and also received soil abatement at Phase 2. These effects were all statistically significant (even "highly significant" by conventional standards of statistical confidence).

The sensitivity of these results was tested by several different methods of statistical analysis. Outcomes based on repeated measures analysis of variance were not sensitive to methodology, being quantitatively similar in other methods, such as longitudinal structural equations models.

To test the response of subsets of children, the data were stratified by age, race, gender, and initial blood lead. There were some differences in blood lead response between boys and girls. In Phase 1, there were larger and more significant responses to soil abatement among boys than girls, whereas in Phase 2 there was a somewhat larger effect among girls in the treatment group that received both dust abatement in Phase 1 and soil abatement in Phase 2. There was also a suggestion in repeated measures analyses of covariance of some differences in either blood lead responses or environmental lead pathways in residences with boys versus girls, but the sample size was too small to allow much exploration of these interesting hypotheses. There were also large effects of soil lead abatement and dust lead abatement identified in Afro-American children, possibly related to differential responses to dust lead, but the sample size did not allowed more detailed exploration of these hypotheses. While there were some large reductions in blood lead in

some younger children who started the study at ages 9 to 17 months, there were not enough of these younger children in the Boston study to show statistically significant effects in this age group, even though the estimated size of the effects was sometimes similar to that seen in the largest age group, 18 to 41 months. Soil abatement effects were somewhat smaller in children older than age 42 months, although this group also had a much smaller sample size than the middle group.

Pre-abatement blood lead concentrations were truncated to the range 7 to 24 μg/dL in the Boston study. The upper limit was imposed by a Massachusetts requirement that children with blood lead levels of 25 μg/dL or more be referred to authorities for treatment or intervention; thus, such children could not be retained in the study lest they be given medical treatment or environmental interventions not assigned in the study design. The lower truncation limit was imposed because of the concern of the Boston investigators that changes in blood lead in children with blood lead less than 7 μg/dL might be too difficult to detect. After discussion with EPA staff, external reviewers, and staff of the other study teams, the decision to truncate the range of blood lead values was accepted as appropriate. Nonetheless, EPA has evaluated possible effects of blood lead truncation in this report. The Boston data were reanalyzed here using a number of additional truncation subsets. Each further truncation of the data reduced the sample size, which generally reduced the statistical significance of the estimated effects. In most cases, the magnitude of the effects remained the same or similar following each truncation. For children with initial blood lead levels of at least 10 μg/dL, the magnitude of the effect and its statistical confidence, increased. In general, it does not appear likely that the findings of the Boston study would have been very different if a truncation different from 7-24 μg/dL had been used.

There was, however, another indication that the blood lead truncation may have had some effect, even though it did not alter the conclusions from Boston as a longitudinal intervention study. Cross-sectional structural equation models were used here to assess the initial relationship of preabatement blood lead, dust lead, and soil lead in order to investigate environmental pathways before abatement. While there was a strong relationship between floor dust lead, window dust lead, and soil lead (as has been found in many analogous studies), the relationship of environmental lead to blood lead was relatively weak in Round 1 of the Boston Study. However, the relationship between blood lead and environmental lead

found after abatement, using repeated measures analyses of covariance and longitudinal structural equation models, was quite strong, even in the control group. Our hypothesis is that the study design, which selected residences with high soil lead but with no children whose blood lead exceeded 25 μg/dL, may have weakened the initial relationship between blood lead and environmental lead. It is possible that a group of children who were lead-burdened and potentially very responsive to abatement were excluded by this unavoidable requirement. If this is true, the Boston study might have shown even larger effects if such children had been included.

Thus, while the findings of the Boston study about the effects of soil and dust abatement certainly appear to be valid (and may have underestimated the effects of abatement), these findings should not be used to draw inferences about the entire population of Boston children, since the study was not designed as a representative cross-sectional population study. Neither should the Boston study be used to infer that abatement was either equally effective or not effective at soil lead concentrations less than 1000 μg/g, since no such residences were included. While it would be reasonable to infer that remediation of yards with soil lead less than 1000 μg/g may have a positive but quantitatively smaller benefit for children residing there, the Boston study neither proves nor disproves such an inference.

Finally, the Boston study design is the only example of a randomized experimental design among the three studies. Neighborhood-level differences that were presumably controlled by randomly assigning treatments across neighborhoods were not similarly controlled in Baltimore or Cincinnati. The advantages of the randomized experiment may have facilitated detection of effects in the Boston study, even in the face of limitations imposed by blood lead truncation.

5.7.2.2 Cincinnati Study

Unlike the Boston study, in which there were indications of changes in environmental lead exposure or environmental dust lead pathways in the soil lead and dust lead abatement groups, there were no substantial indications of any such effects in the Cincinnati study. While the Cincinnati study showed clear differences among neighborhoods, the differences were not aligned with soil or dust abatement, nor were they attributable completely to

differences in soil lead or floor dust lead. Window dust lead was, however, an important contributor to floor dust lead, which was a statistically significant predictor of blood lead in the best-fitting Cincinnati models. There were also strong relationships between floor dust lead and other interface media, such as lead at the interior entry of the residence unit. It is likely that lead from another external source was a contributing factor in lead exposure for children in some neighborhoods. Unfortunately, the "control" neighborhood of CIN NT(M) appears to be one of these, and some children in the Phase 1 soil abatement neighborhood of CIN SEI(P) may also have been affected. An essential requirement of an intervention study is that the effects of important factors that could affect the outcome of the study be controlled by design, by randomization, or by stratification and covariate adjustment. It is not clear that the Cincinnati study met this requirement.

Among the largest and most significant differences in the study is the difference in blood lead response between the two no-treatment groups. This difference suggests that significant factors other than soil abatement were affecting blood lead concentrations in Cincinnati. These factors have not yet been identified by analysis or via other information. Soil abatement in CIN SEI(P) in Phase 1 appeared to have a positive effect compared to one of the groups, CIN NT(M), but a negative effect compared to the other treatment group, CIN NT(G). These inclusive findings emphasize the difficulty in identifying and maintaining appropriately matched control groups in neighborhood-level environmental intervention studies.

5.7.2.3 Baltimore Study

The Baltimore study showed virtually no effect from soil lead abatement. While blood lead in some children in the soil abatement group decreased substantially, there were also decreases in some children in the control group in the other neighborhood. Likewise, some of the children in the soil abatement group and in the control group showed large increases in blood lead after the soil abatement occurred. Several factors appear to be associated with these findings:

1. The Baltimore study was the only study that did not carry out interior dust abatement or interior paint stabilization, and many homes had large concentrations of lead in interior dust and paint;

2. Soil lead concentrations in the yards around many of the residences were relatively low when measured on a yard-average basis, even though for nearly all homes there was at least one location in the yard with soil lead greater than 500 μg/g;

3. All of the remediated housing was located in one neighborhood, and most of the non-remediated housing in another neighborhood, which may not have allowed adequate control for between-neighborhood differences;

4. No data were available on changes in environmental lead concentrations after abatement in the control groups in either neighborhood.

There is little indication that the soil lead abatement substantially or persistently reduced childhood lead exposure in Baltimore. In view of the large quantity of lead in interior dust and paint in most residences, it is likely that this unremediated reservoir of exposure continued to affect blood lead in children after soil abatement was carried out. While soil abatment and exterior paint stabilization may eventually cause reductions in the component of interior dust lead concentration attributable to exterior sources, it did not appear to do so during the time frame of this study to an extent that was detectable. This is not too suprising in view of the probable ongoing recontamination of the dust from interior sources such as paint and from unremediated exterior sources such as resuspended surface soil from nearby residences. The environmental data collected in the study were not adequate to identify such processes, however.

The design of the Baltimore study cannot preclude differences or changes in neighborhood-level lead exposure that may also have been important, such as was observed in Cincinnati. While there was a control group of houses in Area 1 (BAL P2) that were not remediated, all but two had no soil samples with lead concentrations above 500 μg/g and therefore were not comparable to the remediated houses which all had at least one soil sample above 500 μg/g. Additional analyses of pre-abatement data to identify differences between the neighborhoods may be useful.

5.7.3 Summary of Results

The data presented in this section lead to the following conclusions:

(1) Soil abatement in each study effectively reduced the concentration of lead in the soil in the areas where soil abatement was performed.

(2) In the Boston and Cincinnati studies, the effectiveness of soil abatement was persistent through the end of the study. There were no followup measurements of soil in Baltimore to demonstrate persistency.

(3) Reductions of dust lead due to exterior dust abatement, performed only in Cincinnati, were not persistent, indicating a source of lead other than soil at the neighborhood level.

(4) Hand lead measurements often reflected general trends in blood lead measurements and may be a reasonable estimate of recent exposure. Hand lead, as measured in these studies, can be a useful complement to blood lead measurements.

(5) Paint stabilization as performed on all homes with lead-based paint in Boston (interior) and Baltimore (exterior), was intended to reduce the potential confounding effects from contamination of soil and dust, but in retrospect, paint stabilization itself represents one form of intervention in this study.

(6) The Boston study may have also affected blood lead concentrations in the soil lead and dust lead abatement groups, either by modifying exposure (as suggested by changes in blood lead versus dust lead regression coefficients) or by changing soil-to-dust pathways (as suggested by structural equation models). These changes are additional possible effects of abatement beyond the persistent reduction in soil lead and dust lead concentrations.

(7) There was little evidence of changes in dust lead exposure or in dust lead pathways in the for the soil abatement neighborhood in the Cincinnati study, based on longitudinal ANCOVA or structural equation models.

(8) Changes in blood lead in the Cincinnati study were associated with changes in dust lead, but soil abatement was not effective in reducing dust lead compared to changes in some other groups in this study.

(9) There was a marginal indication that blood lead reduction was greater in the Baltimore soil abatement group than in the small control group in the same neighborhood that was not abated since soil lead concentrations were low, but no indication of benefit compared to a control group in another neighborhood.

(10) Based on the Baltimore and Cincinnati studies, there appear to be some relatively large differences in neighborhood-level changes in lead exposure in these urban areas that may constitute a major source of variability in response to soil or dust abatement.

(11) Assessments of the Boston data suggest that some differences may exist between boys and girls in their response to soil abatement, which may be related to age or behavior.

(12) Children in the age group 18-41 months showed the greatest reduction in blood lead from soil abatement in the Boston study, about 2.5 ug/dl in Phase 1. The effects were larger in Phase 2. While there seemed to be a large effect of soil abatement for younger children, it was not statistically significant in the Boston study due to the small number of children less than 18 months of age at the time of abatement.

(13) Blood lead reduction of about 1.9 ug/dl associated with soil and interior dust abatement occurred during Phase 1 of the Boston study, and persisted into Phase 2 with no further abatement.

(14) Soil abatement during Phase 2 of the Boston study was associated with a reduction in blood lead 2.0 ug/dl in the group that received only paint stabilization in Phase 1, and with a reduction of 3.8 ug/dl in the group that received interior paint stabilization and dust abatement in Phase 1, compared with the group that received no Phase 2 abatement.

(15) While the initial truncation of blood lead range in the Boston study may have attenuated the initial relationship of blood lead to soil lead, truncation had little effect on the final results.

5.7.4 Limitations of the Statistical Methods

The statistical methods used here were reasonable and appropriate, and could be used by other investigators with access to standard statistical software packages. However, the methods have certain limitations that should be understood. The repeated measures analyses assume only that the response variables are correlated with each other, with no implication of temporal causality. The goodness of fit of the models was significantly improved by use of covariate analyses.

A problem arises if the response variable must be transformed (e.g., by a logarithmic transformation for blood lead or for hand lead) in order to reduce skewness and to stabilize variances across treatment groups. The implied model for the original untransformed variable is then *multiplicative* in treatment effects and random variation. This is probably acceptable for the analysis of variance, but is likely to produce a physically or biologically meaningless specification for the covariate model when the covariates are indicators of

distinct and additive sources of lead, such as soil lead and interior lead-based paint. The logarithmic model does not reproduce the *additive* nature of the separate exposure pathways.

Extension of repeated measures analyses to covariates such as environmental lead levels that change with time can be done using a single technique, structural equation modeling. These methods provide more powerful interpretive tools. The availability of environmental data to characterize time-varying lead exposures in the Boston and Cincinnati studies suggests that more powerful statistical methods, such as structural equation models, may be more appropriate.

There were substantial differences in the design of the three studies that precluded completely identical analyses of the data. It was technically possible to create a combined data set, given that all three studies included data on blood lead and hand lead before and after abatement, carefully coordinated measures of family demographic characteristics, and both soil and dust lead at the child's residence. Also, some parameters are the same across studies, such as the persistence parameter for blood lead used in structural equation models. However, the substantial differences in study design, such as the characterization of the "control" groups, pre-abatement paint stabilization, age distribution at the time of abatement, ethnic and racial characteristics of the populations, and pre-abatement soil lead exposure meant that mathematically similar measures of effect in each study would have very different interpretations, and would not be clearly generalizable to other study designs, much less to soil lead abatement in other communities. Therefore, no "combined" analyses of pooled data from all three studies was attempted here.

5.7.5 Comparison Across the Three Studies

The effectiveness of soil lead abatement in reducing blood lead varied greatly among the three cities. The variability in abatement effects is probably due to substantial differences in lead sources and pathways among the neighborhoods in these studies. These differences for each study are discussed below.

The Baltimore study had two neighborhoods, Lower Park Heights and Walbrook Junction. The area to which abatement was assigned (Park Heights) had enrolled some families whose residences did not have soil lead levels that were high enough to justify abatement. The nonabatement houses in Park Heights were used as an additional control

group. Unlike the other two studies, the soil abatement in Baltimore was not accompanied by interior dust abatement. There was essentially no significant effect of soil abatement in the abated houses, compared to the control group. It is likely that interior paint contributed to child lead exposure, either directly by ingestion of paint chips, or indirectly by the hand-to-mouth exposure pathway, as follows:

interior paint ⇒ interior dust ⇒ hands ⇒ blood.

Cross-sectional and longitudinal structural equation analyses could be used to explore this hypothesis. However, because there were no repeated measurements of household dust lead, it was not possible to assess changes in exposure over time except by use of hand lead data. Concerning the Baltimore study, we conclude that:

> *It is likely that soil lead abatement had little effect on the primary factors contributing to elevated pediatric blood lead levels in these two neighborhoods; those factors appear to be interior lead-based paint and interior dust lead.*

The Boston study was conducted with blood and hand leads measured at one preabatement round and at about 8 months after abatement. Soil and dust lead measurements were available for pre- and postabatement at about the same time. These data allowed a very complete analysis of blood lead responses to changes in dust and soil lead over time. Relative to the no treatment group, the results showed clearly that there was a persistent reduction (1 to 1.9 μg/dL) in average blood lead levels for the soil lead abatement children and that, on average, the postabatement blood leads were lowest in premises that had the lowest postabatement soil lead and dust lead loadings. Interior and exterior lead paint were not significant predictors of blood lead for Boston children. Concerning the Boston study, we conclude:

> *When soil and dust lead levels show a persistent decrease as a result of effective abatement, blood lead levels also show a persistent decline.*

Because the Cincinnati study had collected blood lead and environmental samples in six Cincinnati neighborhoods, analyses comparable to those reported for the Baltimore and Boston studies can be made. After some analyses using models similar to those for Baltimore and Boston, it became evident that the neighborhoods within each of their treatment group were not comparable in every way. Although there was a strong dependence

of blood lead on environmental lead, particularly on hand lead and on current floor or entry dust lead there was no clear pattern of change or response of interior dust lead levels after abatement.

We are inclined to accept the conclusion of the Cincinnati investigators that blood and dust lead levels were affected differently at different times and places by other events not under their control. However, the dose-dependence exhibited in the models suggests that reducing interior dust lead levels did reduce blood lead levels, at least for a while. The problem is that the abatements did not always persistently reduce dust lead levels. We therefore conclude that:

> *There were additional sources of environmental lead exposure that had different effects on the neighborhoods during the course of the Cincinnati study and were not related to the abatement methods used in the study. It will be necessary to use other analysis methods, such as structural equations modeling, in order to determine the extent to which changes in Cincinnati child blood lead levels may have occurred in response to changes in lead exposure.*

6. INTEGRATED SUMMARY AND CONCLUSIONS

6.1 PROJECT OVERVIEW

This project focuses on the exposure environment of the individual child, looking at three indicators of exposure: blood lead, hand lead, and house dust lead. From the perspective of the child's environment, changes in the soil concentration are expected to bring about changes in the house dust concentration, the hand dust loading, and the blood lead concentration.

In the past 25 years, concern for children with lead poisoning has steadily increased with mounting evidence for the subtle but serious metabolic and developmental effects of lead exposure levels previously thought to be safe. Childhood lead poisoning was formerly considered an acute medical problem usually traced to swallowed chips of peeling lead-based paint. Scientific evidence has systematically revealed deleterious effects of lead from several sources at lower levels of exposure. Agencies such as the U.S. Environmental Protection Agency and the Centers for Disease Control and Prevention (CDC) have repeatedly lowered the level of concern for children's lead burden that recommends environmental or clinical intervention from a blood lead level of 30 μg/dL established in 1978 by CDC to 25 μg/dL in 1985, just prior to the start of the project, then to the present level of 10 μg/dL, which was defined in October 1991 by CDC as a blood lead level that should trigger community-wide prevention activities if observed in many children.

The purpose of Urban Soil Lead Abatement Demonstration Project (USLADP) was to determine to what extent intervention in the form of soil abatement in residential neighborhoods would be effective as a means to reduce childhood lead exposure. Each of the three studies in the project is a longitudinal study of the impact of intervention on the lead exposure of children. The studies focused on evaluation of the exposure environment of the children living mainly in inner city neighborhoods. Measurements of lead in key external environmental media (e.g., soil, exterior and interior dust, and paint) were obtained prior to soil abatement, along with more direct indices of personal exposure in terms of hand wipes and blood lead levels. Abatement of soil lead generally involved removal of contaminated soil and replacement with clean soil. Postabatement lead levels in the above media and

children's blood lead were remeasured at varying intervals to determine the effect of soil abatement, alone or in combination with paint stabilization or dust abatement, on blood lead concentrations. There are few other longitudinal studies of this type, and none of this scope or duration. Because the three studies were conducted using mutually agreed upon protocols, with few exceptions, a common ground exists for understanding an array of information available from the three individual studies that broadens the base of information beyond the limits of a single study or location.

Although the three studies were conducted independently, an effort was made to coordinate the critical scientific aspects of each study in order to provide comparable data at their completion. This effort included seventeen workshops where the study designs, sampling procedures, analytical protocols, and QA/QC requirements of each study were discussed with a goal toward reaching a common agreement. In most cases, a consensus was reached on the resolution of specific issues, but the individual studies were not bound to conform to that consensus or to adhere to it throughout the study. This procedure produced similar studies with some differences in study design and experimental procedures.

The individual results for each of the three cities were originally presented at an EPA-sponsored symposium in August 1992. These presentations included the data analysis and conclusions for each of the three individual city studies. Following this open discussion with the scientific community, the three research teams submitted their respective reports to the designated EPA regional offices (Boston, Region I; Baltimore, Region III; and Cincinnati, Region V). These reports and their associated data sets were then provided to EPA's Office of Research and Development (ORD) and Office of Solid Waste and Emergency Response (OSWER) for further analysis and preparation of this Integrated Report.

The EPA review of the study designs, chemical analytical procedures and data quality measures has found no major flaws that would cast doubt on the findings of the individual reports. The data sets submitted to EPA were systemically scrutinized for errors and inconsistencies, and were reviewed and revised by the principal investigators for each of the three cities prior to the completion of the analyses reported here. These corrections were minor and would not have altered the conclusions of the individual city reports.

This EPA Integrated Report has reached its present form after an extensive review process. First, the reports of the individual studies were peer reviewed by non-EPA experts.

revised, and presented to EPA in their final form, along with the data sets that were used as the basis for the individual reports. These data sets were then reanalyzed by EPA using rigorous statistical techniques to extract information not easily accessible from any individual study. Earlier drafts of this report, based on those analyses, have undergone several rounds of internal and external review. This has included release of the report in draft form for public comment and external review at two separate expert workshops. Further statistical analyses (based in part on peer review comment recommendations) have since been carried out, and the report incorporated changes reflecting the new analyses and earlier comments from the external experts. However, due to time constraints and other factors, it has not been possible to carry out the entire range of analyses that may have been desirable to more fully address important and interesting issues related to the interpretation of findings from the subject studies.

Electronic copies of the underlying three cities data sets will be made available to members of the scientific community for continued review and analysis along with the release of the final version of this report. This continuing reanalysis means that new perspectives on the USLADP data may emerge. Although it is unlikely that major findings have been overlooked during the above-noted extensive review phases, it is not at all unreasonable to expect that still further information will be retrieved and reported by further evaluation to be made possible by this open policy for data release.

6.2 SUMMARY OF FINDINGS

6.2.1 Comparison of EPA Integrated Report Results with Individual Study Results

This integrated assessment looks at the three individual studies collectively to determine if a broad overview can be taken of the project results when each study is placed in its correct perspective.

6.2.1.1 Boston Study

The key findings of this integrated assessment with regard to the Boston study are as follows:

1. The median preabatement concentration of lead in soil was relatively high in Boston, averaging about 2,400 μg/g with few samples below 1,000 μg/g.

2. Abatement of the soil effectively reduced the median concentration of lead in the soil to about 150 μg/g (an average decrease of about 2,300 μg/g).

3. Soil was clearly a part of the exposure pathway to the child, contributing significantly to house dust lead.

4. Other sources of lead, such as interior lead-based paint were minimized by stabilization.

5. The reductions of lead in both soil and house dust persisted for at least two years.

6. Blood lead levels were reduced by approximately 1.9 μg/dL at 10 mo after soil lead abatement.

7. Additional reductions in blood lead of about 2.0 μg/dL (relative to non-abated) were observed at 22 mo postabatement for children in houses where the soil lead was abated and the interior house dust lead was consequently reduced and remained low.

The Boston study used analysis of variance methods based on blood lead differences, and analysis of covariance methods with the longitudinal aspect included by use of the preabatement blood lead concentration (Round 1) as a covariate. The results of their "crude" analysis (Table 15-10 in the Boston study report) are virtually identical to the effect size estimates we calculated for the group as a whole using repeated measures ANOVA and also using a longitudinal structural equations model. Table 6-1 provides a comparison of the results from the Boston individual city report and from this report. The effect size estimates are somewhat smaller in their "base" model, which the longitudinal analysis of covariance model adjusted only for pre-abatement blood lead. In view of the differences in methods and approaches, the overall conclusions are very similar.

The Boston investigators also studied the sensitivity of the effect size estimates to a large number of other covariates, including environmental factors, family demographic factors, behavioral factors, and biological covariates. None of these changed the estimated effect of bos SPI vs BOS P-S (soil abatement vs control) from their base model, 1.49 μg/dL.

TABLE 6-1. COMPARISON OF PHASE 1 EFFECT SIZE ESTIMATES BETWEEN THE BOSTON STUDY REPORT AND THIS REPORT

Group Study		Boston Report[1]		This Report[1]	
Abate Versus Control		Crude Model	Base Model	RM ANOVA	LSEM Model 17
BOS SPI	BOS P-S	1.92	1.49	1.87	1.86
BOS SPI	BOS PI-S	1.53	1.28	1.54	1.56
BOS PI-S	BOS P-S	0.39	0.21	0.33	0.30

[1]Units are μg/dL reduction of Pb in blood.

by more than 0.22 μg/dL. The factors were entered one at a time. The largest decrease was seen with inclusion of race as a factor (which reduced the effect to 1.27 μg/dL) and with inclusion of pre-abatement lead paint (which reduced the estimated effect to 1.34 μg/dL). Five factors decreased the effect size, which nevertheless remained statistically significant: water lead concentration, time away from home, time away from study area, playing or sitting on inside floor, and ferritin level. The other 15 factors tested increased the estimated effect size, particularly age (to 1.61 μg/dL) and hand washing before meals (to 1.63 μg/dL), as well as: gender, socioeconomic status, mouthing variables, chipping paint, yard play, outdoor eating, hand washing after outdoor activity, pets that go outdoors, imported canned food, lead-related occupations, lead-related hobbies, cigarette smoking, and owner occupancy. Many of these factors are important in identifying individual exposure components and lead risk factors, and are worthy of additional scientific investigation. However, none of these factors appear to have interacted so strongly with soil and dust abatement as to have qualitatively affected the conclusions of the study, except for relatively small effects related to age, race, and lead paint level. Much of the lead paint effect is mediated, both statistically and physically, by lead concentrations or loadings in house dust. It is likely that the use of household dust as a covariate in the models of this report effectively subsumed the lead paint effect, and that the dust abatement that was carried out in the Boston study along with soil abatement may have affected some fraction of the blood lead response that might have been otherwise attributed to lead-based paint. Even so, the overall

treatment group effect in the model that included lead paint was only slightly less significant (P = 0.05) than the base model (P = 0.02). On the other hand, including chipping paint in the model increased the effect to 1.53 μg/dL (P = 0.02 for the group model, P = 0.01 for the BOS SPI vs BOS P-S effect). Additional studies involving the paint contribution to the total lead exposure pathways, and assessment of the possible effects and interaction between paint condition and paint lead loading on lead exposure, are needed to understand the relatively small modifications of effect size attributable to lead paint.

Age and race effects are larger than the paint effects and were evaluated in this report. Larger effects were identified for children of ages 18 to 41 months, and for children of Afro-American ancestry, than for the sample as a whole. The Afro-American children also seemed to show larger responses to dust abatement than did the sample as a whole.

In summary, the abatement of soil in the Boston study resulted in a measureable, statistically significant decline in blood lead concentrations in children, and this decline continued for at least two years. It appears that the following conditions were present, and perhaps necessary for this effect: (a) a notably elevated starting soil lead concentration (e.g., in excess of 1,000 to 2,000 μg/g); (b) a marked reduction of more than 1,000 μg/g in soil lead consequent to soil abatement accompanied by (c) a parallel marked and persisting decrease in house dust lead.

These conclusions are consistent with those reported by the Boston research team. This integrated assessment found no basis for modifying their conclusions, although we choose not to express these findings as a broadly generalizeable linear relationship between soil and blood, such as change in micrograms of lead per deciliter of blood per change in micrograms of lead per gram of soil, because we believe that such a linear expression of abatement effects is highly site specific for the soil-to-blood relationship. We found evidence that the dust-to-blood relationship is more significant than the soil-to-blood relationship and therefore the abatement effect also depends on soil-to-dust transfer, which may be very site-specific.

6.2.1.2 Baltimore Study

With regard to the Baltimore analyses conducted for this integrated assessment, the participants in the abatement neighborhood that did not receive abatement were treated as a separate control group, rather than combined with the nonabatement neighborhood (as the

Baltimore research team did). The reason for this was to establish a control group not influenced by differences between neighborhoods. This alternative approach used in this integrated assessment had little impact on the statistical significance of soil abatement effects as reported by the Baltimore research team.

The key findings of this integrated assessment for Baltimore are:

1. The preabatement concentrations of lead in soil were notably lower (i.e., averaging around 500 to 700 μg/g, with few over 1,000 μg/g) than in Boston.

2. The actual reduction of lead in soil by abatement was small (a change of about 400 μg/g), compared to the Boston study (a change of about 2,300 μg/g).

3. Measurements of blood lead were made for only ten months following abatement; and no significant decreases in blood lead consequent to soil abatement were observed compared to non-abatement control group children.

4. Except for exterior lead-based paint, there was no control of other sources of lead, such as the stabilization of interior lead-based paint (as done in Boston) or abatement of house dust (as done in Boston and Cincinnati).

5. Follow-up measurements of soil (except immediately postabatement) were not made to establish the persistency of soil abatement, and its possible effects on house dust.

The Baltimore report used a generalized linear regression model (GLIM). In its simplest form, the regression model can be expressed as a linear model using log-transformed variables. The Baltimore blood lead model 1 is a simple ANOVA model,

$$\text{Log}(BC_{ij}) = G_{gj} + e_{ij}$$

with only two treatment groups, Area 1 and Area 2. However, Area 1 includes some non-abated residences as well the residences that received soil abatement, whereas Area 2 includes only non-abated residences. Therefore, the results in the Baltimore report cannot be directly compared with the results reported here, where we have separated the abated and non-abated residences into two groups and used the non-abated residences in Area 1 as a second control group. Model 2 in the Baltimore report is a simple ANCOVA model,

$$\log(BC_{ij}) = G_{gj} + b_{2j}\,\text{Age}_{ij} + b_{3j}\,\text{SES}_i + b_{4j}\,\text{Season}_{ij} + b_{6j}\,M_{ij}\,\log(\text{Hand}_{ij}) + b_{7j}\,(1-M_{ij})\,\log(\text{Hand}_{ij}) + e_{ij}.$$

In this notation, Age is a semi-categorical variable, Season is included only for preabatement rounds 1 and 2 that covered many months, and M_{ij} is a dummy variable for low or high mouthing behavior. While temporal comparisons are possible, no temporal correlation model is assumed, and the Baltimore report notes that the lack of temporal modeling is a deficiency in the analyses.

The Baltimore analyses were carried out for two distinct subgroups of children. The first set of analyses used only those children who were present in all six rounds. The second set of analyses used all children who were present in each round. Analyses for this report used children who were present in Rounds 3, 4, and 6. The set of children who were present in all rounds is included in the EPA set, but does not include other children in the EPA set such as those children who were recruited at Round 3, especially very young children. The second set of children in the Baltimore study is much closer to the EPA child set in Rounds 4 and 6, but includes in Round 3 some additional children who dropped out after Round 3. Therefore, the EPA effects size estimates are based on different groups of children than in the Baltimore report.

Effect sizes were calculated in Table 6-2 as simple differences of treatment group effects reported in Tables 7-7 and 7-8 of the Baltimore report. The effects were small and probably not statistically significant, although the lack of correlation structure in the Baltimore models makes any estimates of standard errors rather questionable. The differences in blood lead are negative between the treatment group (BAL SP) and the control group (BAL P1 and BAL P2). There is little reason to believe that major treatment group differences would have been identified by other analyses of these data.

Other findings in the Baltimore study are of interest. There were some indications of significant differences associated with hand land, with a modifying effect due to child mouthing behaviors. There was also a strong effect of socioeconomic status on blood lead and dust lead, and an age effect with maximum blood leads at ages 1 to 3 years (12 to 36 months), a general finding in these studies.

Thus, in Baltimore, where the differences between pre- and postabatement soil lead concentrations were much less than in Boston, and where the soil abatement criteria left some properties only partially abated, and where no interior paint stabilization or dust

TABLE 6-2. EFFECT SIZE ESTIMATES FROM THE BALTIMORE REPORT COMPARING BLOOD LEAD REDUCTION IN BAL SP VERSUS CONTROLS

Rounds	Child Group	Baltimore Model[1,2]		This Report[1,3]	
		ANOVA	ANCOVA	BAL SP vs BAL P1	BAL SP vs BAL P2
Rounds 3 and 4	All 6 Rounds	-0.55	0.12	0.07	1.77[4]
	Each Round	-0.07	-0.10		
Rounds 3 and 6	All 6 Rounds	-0.92	-0.71	-0.54	0.67
	Each Round	-1.55	-1.17		

[1]Units are μg/dL reduction of Pb in blood.
[2]Baltimore controls are BAL P1 and BAL P2
[3]Children present in Rounds 3,4, and 6
[4]P=0.16; others, P>0.2.

abatement was performed, no detectable effects of soil lead abatement on blood lead levels were found.

These conclusions are consistent with those reported by the Baltimore research group, and are not inconsistent with those above for the Boston study. At soil concentrations much lower than the Boston study, the Baltimore group would have likely been able to see only a very modest change in blood lead concentrations (perhaps less than 0.2 μg/dL), assuming similarity between the study groups in Boston and Baltimore and the same linear relationship between change in soil concentration and change in blood lead. Furthermore, the interior paint stabilization and house dust abatement performed in Boston perhaps enhanced and reinforced the impact of soil abatement on childhood blood lead, whereas in Baltimore, any possible small impact of soil abatement would have likely been swamped by the large reservoir of lead in the interior paint and the large unabated amounts of lead in interior house dust.

6.2.1.3 Cincinnati Study

As for the Cincinnati study, because of differences in the neighborhoods, we found that combining neighborhoods into treatment groups often obscures important effects, and chose to analyze each of the six Cincinnati neighborhoods as separate treatment groups. One neighborhood, CIN I-SE(B) had an insufficient number of participants and was dropped from

some analyses. The CIN I-SE(B) group started with nine families, but by Round 5 there was only one participating family in the study. The two control neighborhoods, CIN NT(G) and CIN NT(M), were also found to be substantially different, and that the three remaining treatment groups, CIN SEI(B), CIN I-SE(D), and CIN I-SE(F), were more comparable, both demographically and in geographic proximity, to CIN NT(M) than to CIN NT(G).

The Cincinnati study used several different regression (ANCOVA) models, and cross-sectional structural equation models. The report also included results of a simple correlation analysis that did not allow for multiple covariate adjustments, and is not further described. The response variables in the regression models included the difference in blood lead between Round 1 and Round 4, hand lead differences, and differences in interior floor dust loading and in exterior dust loading. The final regression model for the change in blood lead involved only blood lead concentration (which we denote Blood), hand lead loading (which we denote Hand), age of the child at the Round 4 blood lead measurement (which we denote BloodR4), and socioeconomic status (denoted SES). In our notation, their model is:

$$\text{BloodR4} - \text{BloodR1} = 8.52 + 0.038\,(\text{HandR4} - \text{HandR1}) - 0.00079\,\text{AgeR4}*\text{HandR4} - 0.17\,\text{SES} - 0.43\,\text{BloodR1}.$$

This model has one point of similarity to our Cincinnati longitudinal SEM models. By transposing the BloodR1 on the left side of the equation, we have a linear relation that is expressed algebraically as BloodR4 = 8.52 + ... other terms + 0.57 BloodR1, which is close to the value of the blood lead persistence parameter A_{14} obtained for most of the Cincinnati LSEM models, such as $A_{14} = 0.58$ in Model J6 used in the effects size comparisons. Otherwise, blood lead is not predicted by neighborhood, nor by abatement group, nor by environmental lead concentrations or loadings, but by another time-variable and child-specific variable, hand wipe lead loading, which tends to increase with the child's age. The regression model for hand lead change also excludes treatment group or environmental variables, except indirectly through Round 1 hand lead.

The report also presents a structural equation model for blood lead and hand lead differences, and for changes in interior and exterior dust lead. Their equations for blood and hand lead are, in our notation:

$$\text{BloodR4} - \text{BloodR1} = 10.28 - 0.18\ \text{SES} - 0.064\ \text{AgeR5} - 0.46\ \text{BloodR1}$$

$$\text{HandR4} - \text{HandR1} = 5.78 + 0.002\ \text{HandR5} - 0.62\ \text{HandR1}.$$

The two dust lead equations are totally unconnected to blood lead or hand lead.

The report also shows cross-sectional structural equation models for Round 1, Round 3, and Round 4 respectively. The Round 1 SEM model shows large and statistically significant age effects, and effects of mouthing behavior. Areas and neighborhoods show no significant differences. The model uses no environmental covariates, but reports a significant regression of log(BloodR1) on log(HandR1). The simultaneous equation for log(HandR1) depends strongly on age and not at all on treatment group or neighborhood. Neither equation uses any of the environmental covariates, but both include a significant fixed effects factor for "families", which is analogous to the random effects term Hh(g) in our repeated measures ANOVA and ANCOVA models. However, their findings of no significant neighborhood differences or environmental factors differs somewhat from some of the findings in our cross-sectional and longitudinal SEM models. Differences in model format and structure make direct comparisons very difficult.

The Cincinnati investigators concluded that the Phase 1 changes in blood lead concentrations and in hand lead loadings were not significantly different among the three abatement groups, using either multiple regression models or structural equation models. They did not compare across different neighborhoods within treatment groups, which was an additional source of variability in the study. We cannot therefore directly compare our effect sizes or treatment differences across neighborhoods with their aggregated results. Since their models are not directly comparable to our models without additional substantive analyses of the role of hand wipe lead, we cannot directly compare effect sizes using longitudinal SEM.

The Cincinnati report giving a cross-sectional SEM for Round 4 (their Table 4-63) presents a comprehensive and detailed SEM which is in substantial qualitative agreement with the longitudinal SEM we presented for Cincinnati Round 4 blood lead and dust lead. The use of hand lead in their model precludes direct comparisons with the longitudinal SEM shown here in Table 5-39. The use of log(HandR4) as a covariate that is only partially adjusted by window and floor dust lead loadings, age, and SES permits the finding of large,

statistically signficant, but *negative* relationships between log(BloodR4) and dust lead loadings on the floor, interior entry, and exterior. Additional analyses of this model would be useful. The model uses neighborhood or area as an adjustment covariate for hand-to-blood, dust-to-blood, dust-to-hand, paint-to-dust, and exterior-to-floor pathways, with some significant differences. While the application of this model does not allow comparison of effect sizes relative to Round 1, there is a qualitative similarity in our findings with those of the Cincinnati investigators.

On this basis, we concluded that, in most cases, the effect of soil abatement could not be clearly determined, and offer the following explanation for this conclusion:

1. Most of the soil parcels in each neighborhood were not adjacent to the living units, and this soil was therefore not the primary source of lead in house dust. Evidence for this statement includes the observation that street dust lead concentrations are much higher than soil concentrations, indicating there is a large source of lead contributing to street dust in addition to soil lead.

2. The preabatement median soil lead concentrations in the three treatment groups were about 300 μg/g in CIN SEI(P), 700 μg/g in CIN I-SE(F), and 800 μg/g in CIN I-SE(D), and the postabatement soil concentrations were less than 100 μg/g, so that the reduction of lead in soil was small, as in Baltimore.

Evidence for the impact of dust abatement or dust and soil abatement consists of a statistically significant difference between changes in blood lead between Rounds 1 and 4, approximately one year apart. Some Cincinnati neighborhoods showed decreased blood lead concentrations in response to dust abatement or dust and soil abatement. The two neighborhoods that received only interior dust abatement in the first year, CIN I-SE(D) and CIN I-SE(F), showed a small decrease in blood lead concentrations, compared to large increases in the nearest control group, CIN NT(M). The treatment group that received soil, exterior dust and interior dust abatement, CIN SEI(P), showed a smaller effect than did the CIN I-SE(D) and CIN I-SE(F) neighborhoods. After consultation with the Cincinnati research team, we suspect that there was recontamination of street dust in CIN SEI(P) during the study, probably caused by demolition of nearby buildings in the neighborhood.

The consistent theme across the outcomes for all three studies is that soil abatement must be both effective and persistent in markedly reducing soil lead concentrations accompanied by a corresponding reduction in house dust lead in order to result in any detectable reduction of blood lead. The location of the soil relative to the exposure

environment of the child is important. In this project, the movement of lead from soil or street dust into the home seems to be a key factor in determining blood lead concentrations. Although these USLADP results provide substantial evidence for the link between soil or street dust and house dust lead, there is insufficient information by which to clearly quantify this relationship in terms of the lowest level of soil or street dust lead reduction that will yield a measurable decrease of lead in blood.

6.2.2 Synthesis of Findings Across the Three Studies

While the USLADP was not intended to compare different methods for soil abatement, the differences in design and methodology among the three studies helped to identify conditions for which soil abatement may be an effective intervention, and conditions under which soil abatement is less likely to be effective. Abatement or intervention can be effective if it can achieve one or both of the following goals:

1. Abatement or intervention produces an effective and persistent reduction in the concentrations of lead in soil and in household dust.

2. Abatement or intervention changes childhood lead exposure by reducing the intake of lead-contaminated media, or effectively breaks the transport pathway from the lead-contaminated source to the child's activity areas.

These are not mutually exclusive goals, but there are important distinctions among them. The first goal, reducing lead concentrations, can be achieved without changing exposure or transport. For example, removing bare lead-contaminated soil from a yard and replacing it with bare soil that is not contaminated will not immediately change the child's exposure to interior dust lead nor the transport of surface soil from the yard into the house. However, the child's intake of lead directly from any soil ingestion will immediately be reduced, and one would expect that over some period of time, there will be a reduction of the child's intake of lead from household dust because the soil component of household dust lead has been eliminated. All three studies achieved the elimination of lead in yard soil. It is important to note the requirement that the soil not be recontaminated by unremediated sources such as exterior paint and by transport of lead from unremediated areas. Even in the Boston study, a few yards became substantially recontaminated. However, most of the sampled locations in the Boston and Cincinnati did not suffer significantly recontaminated

soil after abatement. The Baltimore sites were not followed up over a similar period of time

Both Boston and Cincinnati residences received interior dust abatement. The Boston residences showed slight evidence of recontamination, whereas most the residences in the areas that received interior dust abatement (with or without soil abatment) during Phase 1 of the study showed significant recontamination. The floor dust lead concentrations showed a significant association with window lead and mat lead, suggesting exterior sources of recontamination. Long-term changes in dust lead were not followed up in the Baltimore study. Significant blood lead reduction was detected only in the Boston study, where persistent reduction of dust lead occurred in most residences that received soil lead and interior dust abatement. The effect was even greater in Phase 2 in the group PI-S that received both Phase 1 dust abatement and both soil and dust abatement in Phase 2.

The second goal, reduction of exposure, requires reducing the amount of potentially lead-contaminated media consumed by the child. Repeated measures analysis of covariance of the Boston study suggests that this may have occurred, based on some statistically significant changes in the regression coefficients between blood lead and dust lead after abatement. Longitudinal structural equation models for Boston also suggest some changes in soil-to-dust or dust-to-blood pathways. Similar analyses of the Cincinnati data find little evidence for changes in regression coefficients. The regression coefficients are generally believed to indicate components of the exposure pathway, either intake of lead-contaminated media by the child or transmission of lead contamination from one medium to another more accessible to the child. Soil abatement can reduce exposure by covering soil with sod or other barriers that reduce the child's access to surface soil particles. The reduction in exposure is distinct from reducing the lead concentration in the soil to which the child is exposed. Likewise, frequent and effective washing or vacuuming of household dust can reduce the amount of dust (dust loading) that is accessible to the child, however much lead is in the dust. Changes in behavior, such as more frequent hand washing or greater parental attention, can also reduce contact with dust and soil. Since all of these studies may have initiated behavioral changes from the moment of recruitment simply by informing parents and caretakers of potential lead hazards, such changes cannot be detected with this study design.

The second goal can also be achieved by any process that reduces transport of the contaminant from the source to the areas in which the child may come into contact with it.

Covering bare soil with sod, concrete, or other barriers will clearly prevent contamination of house dust and outside play areas, just as encapsulation of paint will prevent paint chips from contaminating dust, so long as the barrier remains intact. Removing the source of contamination was shown to be effective in Boston, but in addition to this, there is also some possibility that the post-abatement pathway regression coefficient from soil to dust may have been changed. However, there may also have been a serious attenuation of the apparent pathway in the Round 1 data set, possibly attributable to the blood lead truncation of the study. Additional studies on the effects of soil abatement on environmental lead pathway kinetics would be useful. In general, any method that attempts to estimate post-intervention or post-abatement blood lead concentrations (for example, EPA's IEUBK Model or "slope factor" models) should take into account not only the changes in environmental lead concentrations that may occur as the results of abatement or intervention, but also the changes in the pathways to childhood exposure that may occur following abatement or intervention.

Finally, one should recognize that any environmental lead abatement or intervention may be limited in its ability to reduce blood lead concentrations in currently lead-burdened children. It appears that, in the first year after abatement, at most 40 to 50 percent of the child's previous blood lead burden may be removable by soil abatement or any other combination of abatements and interventions apart from medical treatment by chelation. Thus, there may be a greater effect of lead abatement in preventing lead exposure for future versus current residents, but this possibility cannot be readily assessed, if at all, on the basis of the existing "Three-Cities Lead Study" data sets evaluated in the present report.

6.2.3 Application of Findings to Conceptual Framework of Soil and Dust Lead Exposure Pathways

This integrated assessment attempts to answer the following question: If residential soil is abated will blood lead concentrations decline? To confirm or reject this soil lead/blood lead hypothesis, this report builds a framework of logical arguments described below. Each step of the pathway from soil to blood must be scrutinized closely and related data examined in detail. This means that if dust lead derived from soil is not ingested, either directly or

after passing through other sources, then blood lead concentrations cannot respond to changes in soil lead concentrations.

1. There is a substantial amount of lead in soil.

   Lead was measured in soil in the range of less than 50 $\mu g/g$ to more than 18,000 $\mu g/g$. If a parcel of 100 m^2 had an average of 500 μg Pb/g soil, then the upper 2 cm of soil on this parcel (about 4,000,000 g) would contain 2 billion μg or two kilograms of lead. Before abatement, there was an estimated 25,000 kilograms of soil lead on the participating properties of this project.

   A 2-cm soil core was deemed better than a 15-cm core commonly used in previous studies. When there is a decreasing gradient between the top and bottom of the 15-cm core, the effect is to dilute the concentration, giving a distorted picture of what is available at the surface. In this project, some measurements were made of the soil concentration in the bottom 2-cm of the 15-cm core in order to determine the depth of excavation. The Boston study reported there was not a large gradient between the top and bottom of the 15-cm core, as had been expected.

   Finally, there is little information on the types of surfaces that a child plays on. If these surfaces are mostly soil, as opposed to asphalt or concrete, then the soil measurement may be a good estimate of exposure. However, exterior dust is probably a better estimate of exposure from hard play surfaces (item 5 below). Exterior dust represents lead from several sources, including soil, and may also be a better estimate of the lead transferred to household dust.

2. Lead in soil must be connected by environmental pathways to other compartments of the child's environment, such as exterior dust.

   Limited evidence for this statement was shown in the Cincinnati study. In the Cincinnati study, the relationship between soil and exterior dust was found to be very weak, giving rise to the next statement.

3. There are sources of lead other than soil that contribute to exterior dust.

   Because the changes in lead in soil do not account for all of the changes in exterior dust, it is reasonable to conclude from the Cincinnati study that there are other sources for lead in exterior dust. In Cincinnati, the soil parcels were generally not on the individual properties of the participating families, as was the case in Boston and Baltimore. There are no measurements of exterior dust in the Boston or Baltimore studies.

4. Lead in exterior dust can also move into other components of the child's environment, such as interior dust.

   In the Cincinnati study, when exterior dust lead concentrations changed, interior dust lead concentrations also changed. This was especially obvious when the

exterior dust sample closest to the residence was compared to the interior floor dust sample taken just inside the entryway door.

A living unit with 130 m^2 of floor space (1,400 ft^2) and 1,000 μg Pb/m^2 (a relatively high value from tables in Section 3.3) would have 130,000 μg of lead, or less than 0.01% of the lead available from soil in paragraph 1 above (see Figure 6-1). Additional lead would be in rugs, upholstered furniture, and window areas.

5. There are sources of lead other than exterior dust that contribute to interior dust.

   Taken individually, none of the studies decisively demonstrated this effect. The most obvious source of lead inside the home is lead-based paint, which was common in the Boston and Baltimore studies, but less important in the Cincinnati study. Because neither Boston nor Baltimore measured exterior dust, measurements of interior dust in these studies cannot easily be broken down into contributions from lead-based paint and from exterior dust. However, structural equation analyses on the Boston study showed a strong influence of both interior and exterior lead-based paint on interior dust.

6. Lead in soil can move directly onto the child's hand.

   Conceptually, the transfer of lead from soil to the child's hand is difficult to measure. A child playing outside usually gets soil on his/her hands, but it is not certain whether this soil is adequately represented by a composite of 2 cm soil cores.

7. Lead in exterior dust can move directly onto the child's hand.

   There is no portion of these studies that directly measures this effect. Baltimore reported that the lead loading on hands increased during the summer months, by inference due to the increased playtime outside. During the interviews with the family, questions were asked in all three studies about the activity patterns of the children, including the amount of time spent outside. In the Cincinnati study, the child was observed during the interview period and the handwipes were taken at the end of the interview.

8. Lead in interior dust can move directly onto the child's hand.

   In most cases, when interior dust changed, hand dust changed. Because hand dust lead is only a measure of the amount of lead on the hand, not the concentration nor the amount of dust, it is difficult to make a quantitative estimate of this pathway. It is not likely that the amount of dust on the hand is strictly a function of the amount of dust on the playing surface, as there is probably an equilibrium effect where some dust falls off after time, depending on such factors as moisture content of dust and soil, and conditions on the hand surface. There is no aspect of these studies that could measure this interesting problem.

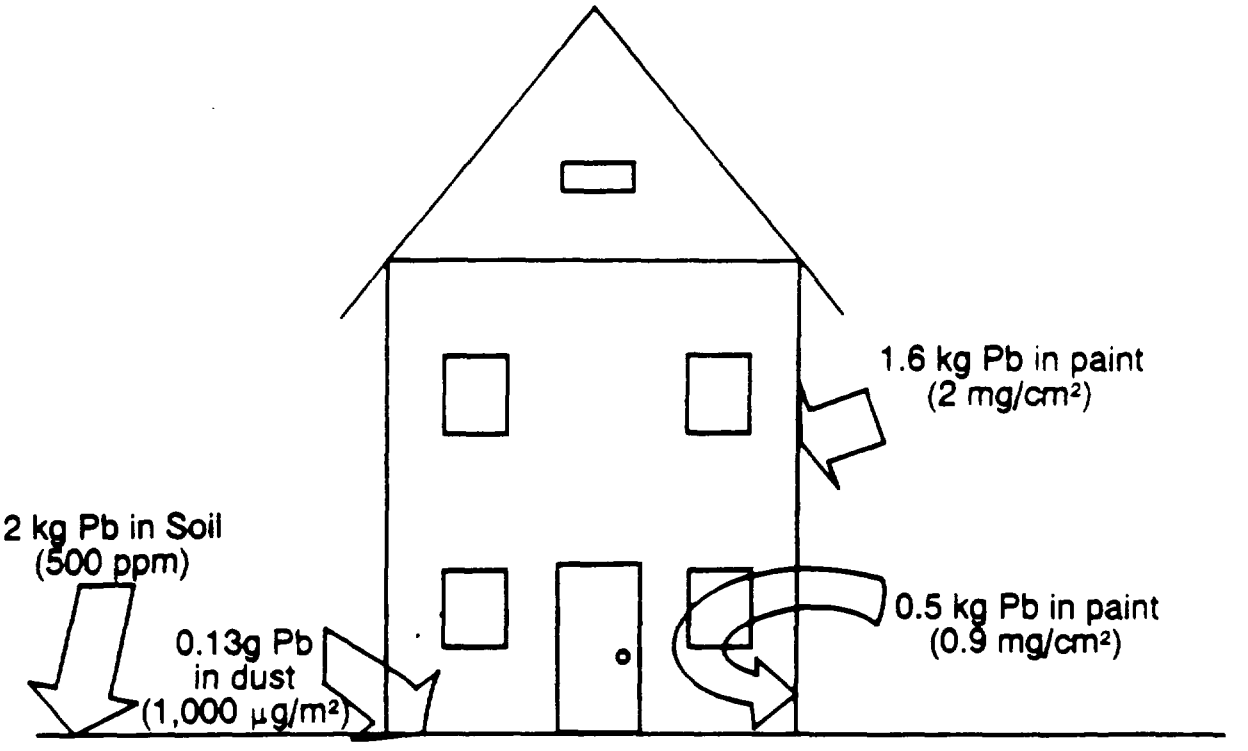

Figure 6-1. Total amounts of lead in various compartments of a child's environment, using the assumptions for concentration (soil, top 2 cm) or lead loading (dust and paint) in parentheses. Although house dust is only a small fraction of the total lead in the child's environment, it is the most accessible component. The concentrations and loadings are illustrative, not typical.

9. Lead in interior dust can also move into other components of the child's environment, such as food.

   This pathway was not investigated by any of the three studies. Measurements of lead in food before and after kitchen preparation would be required. Conceptually, this lead and other routes such as the direct mouthing activities on toys, furniture, and window sills is included in the measurement of interior dust when the assumption is made that a child ingests about 100 mg dust/day by all routes and through all activity patterns.

10. There are sources of lead other than dust that contribute to the child's lead exposure.

    In this project, lead was measured in drinking water once or twice during each study. Low ambient levels (ca. 0.1 $\mu g/m^3$) of lead in air (typical of U.S. metropolitan areas in 1990) were assumed, as were national averages of lead in food. Ethnic food preferences and individual use of cosmetics or other lead containing products were not investigated.

6.3 INTEGRATED PROJECT CONCLUSIONS

The main conclusions of this Integrated Report report are two-fold:

(1) *When soil is a significant source of lead in the child's environment, under certain conditions, the abatement of that soil will result in a reduction in exposure that will cause a reduction in childhood blood lead concentrations.*

(2) *Although these conditions for a reduction in blood are not fully understood, it is likely that five factors are important in determining the magnitude of any possible reduction: (1) the past history of exposure of the child to lead, as reflected in the preabatement blood lead; (2) the initial soil lead concentration the magnitude of the reduction in soil lead concentrations; (3) the initial interior house dust lead loading and the magnitude of the reduction in house dust lead loading; (4) the magnitude of other sources of lead exposure, relative to soil; and (5) the strength of the exposure pathway between soil and the child relative to other lead exposure pathways in the child's environment.*

The basis for the first conclusion is: in Boston, where the soil lead concentrations were high and the contribution from lead-based paint was reduced by paint stabilization, there was a measurable reduction of blood lead concentrations. This reduction continued to increase for two years following abatement in Boston.

Conversely, in Baltimore and Cincinnati, where soil was not a significant source of lead relative to other sources, there was no measurable reduction of blood lead except in cases where those sources were also removed or abated. In Baltimore, these sources may have been interior lead-based paint that was not stabilized, or house dust that was not abated. In Cincinnati, the principal source of lead seemed to be neighborhood dust that may have been contaminated with lead-based paint.

The basis for the second conclusion is: in those cases where all important elements of the exposure pathway were available for assessment, the structural equation model analyses showed that preabatement blood lead concentration was a major predictor of postabatement blood lead, suggesting that the remobilization of bone lead is a major component of the measured blood lead.

All other factors being equal, the measurable reduction in blood lead was observed only at higher concentrations of soil lead. In the absence of information about other sources of lead, no clear statement can be made about the possibility of smaller reductions in blood lead at lower soil lead concentrations.

In spite of the recent successes in reducing exposure to lead by removing lead from gasoline and canned food, lead exposure remains a complex issue. This integrated assessment attempts to assess exposure to lead in soil and house dust. Lead in soil and lead-based paint are closely linked in the child's environment. If there is exterior lead-based paint, then soil lead is likely to be elevated with a consequent elevation in house dust lead. If there is interior lead-based paint, then efforts to reduce the impact of soil lead on house dust will be only partially effective. The maximum reduction in lead exposure will not be achieved unless both paint and soil abatement are implemented.

There is evidence from all three studies that lead moves through the child's environment. This means that lead in soil contributes to lead in street or playground dust, lead in exterior paint contributes to lead in soil, and lead in street dust contributes to lead in house dust. A more detailed analysis of the data may show the relative contribution from two or more sources, but the present analyses imply that this transfer takes place.

The analysis of the data from the three studies showed evidence that blood lead responds to changes in house dust lead. There is also evidence for the continued impact of other, independent sources following abatement of one source. This means that abatement of soil or exterior paint does not necessarily reduce the contribution of lead from other sources such as interior lead-based paint.

The conclusions of this report suggest that soil abatement can have a measurable effect on reducing exposure to lead if there is a substantial amount of lead in soil and if this soil lead is the primary source of lead in house dust. In such cases, both soil abatement and interior dust removal should be performed to be fully effective. In addition, if soil abatement is carried out, then paint abatement should also be considered, where appropriate, to lessen the probability of recontamination of soil and/or house dust. Likewise, soil abatement should be considered in conjunction with paint abatement when it is likely that soil will otherwise continue to contaminate house dust after a paint abatement is completed.

From one perspective, decisions about soil abatement need to be made on an individual home basis. This report shows that, on an individual house basis, soil abatement may potentially reduce the movement of lead into the home and its incorporation into house dust. The magnitude of this reduction will depend on the concentration of lead in the soil, the amount of soil-derived dust that moves into the home, the frequency and methods of cleaning

in the home and the cleanability of the home. The number and ages of children and the presence of indoor/outdoor pets are factors known to increase rate of dust movement, whereas frequent cleaning with an effective vacuum cleaner, use of entry dust mats, and removing shoes at the door serve to reduce the impact of soil lead on house dust.

From another perspective, soil abatement at the neighborhood level poses problems not pertinent to individual homes. Playground, vacant lot, and other plots of soil may pose an immediate problem if they are accessible to children and there is a direct pathway for dust generated by this soil to enter the home. Likewise, sources of lead other than soil may contribute more to exterior dust than soil itself. The evidence in this report suggests that the key to reducing lead exposure at the neighborhood level is to abate significant sources of lead contributing to exterior dust, in addition to the soil and paint abatement that would be performed on an individual property.

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APPENDIX A:

GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

The data in Table A-1 were derived using the PROC UNIVARIATE feature of SAS 6.10 (SAS, 1994). The treatment groups are as described in Chapter 5, using data identical to that plotted in Figures 5-8 through 5-32. Data for blood lead concentration and hand lead are calculated with one value for each child; for floor and window dust, one arithmetic mean value for each living unit; and for soil, one arithmetic mean value for each property or soil parcel. The group assignments and numbers of individuals are different from the individual study reports and different also from the summaries of these reports in Chapter 4. In particular, the data are different from Tables 4-2 through 4-4.

TABLE A-1. GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Boston	Soil Pb Conc.	BOS SPI	1	35	1485	1678	2413	3367	4020	2625
	(μg/g)		2	26	83	98	125	160	190	139
			3	35	50	70	113	192	380	234
			4	21	83	100	174	284	297	206
		BOS PI-S	1	36	1469	1813	2477	3300	4400	2831
			3	35	1460	1480	2148	3286	3833	2502
			4	22	88	161	278	505	570	429
		BOS P-S	1	30	1355	1611	2268	3890	4064	2728
			3	30	1493	1572	2115	3880	4240	2679
			4	17	50	110	204	240	350	307
	Floor Dust Pb Conc.	BOS SPI	1	40	1087	1152	2420	4662	9775	6682
	(μg/g)		2	38	611	657	974	1867	3112	3203
			3	31	553	692	876	1383	2409	1291
			4	28	550	619	726	1182	1568	1239
		BOS PI S	1	39	1256	1429	2582	4380	5764	4278
			2	34	669	865	1302	1568	1960	1481
			3	32	749	870	1198	1591	1902	1373
			4	27	400	517	806	1450	2500	1192
		BOS P S	1	33	1060	1441	2536	4496	8599	5334
			2	35	692	807	1130	1660	3228	1525
			3	32	922	1170	1504	2063	2743	1948
			4	21	550	644	862	1250	1485	1041
	Floor Dust Load	BOS SPI	1	40	9.09	11.24	23.56	69.86	81.01	51.03
	(mg/m^2)		2	38	11.16	13.33	22.78	62.00	94.59	53.14
			3	34	6.89	8.15	15.20	29.76	45.88	25.63

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Boston	Floor Dust Lead		4	28	17.36	24.15	31.29	60.35	88.87	50.90
	(mg/m^2)	BOS PI-S	1	40	9.87	10.80	24.39	41.85	66.96	38.86
			2	34	7.75	14.57	27.06	47.86	69.13	41.31
			3	32	5.37	9.38	17.17	37.77	44.95	27.00
			4	21	16.12	17.01	31.00	50.10	54.56	37.22
		BOS P-S	1	33	9.92	15.25	39.68	70.68	87.63	46.97
			2	32	9.28	11.78	32.24	55.80	94.24	46.95
			3	29	7.79	10.34	18.35	35.09	50.84	28.75
			4	21	9.92	13.33	36.85	76.88	86.30	55.92
	Floor Dust Pb Load	BOS SPI	1	40	26.41	34.94	59.82	124.26	181.43	349.48
	($\mu g/m^2$)		2	38	7.84	12.76	26.48	73.33	192.96	244.39
			3	31	4.51	7.60	18.27	30.33	82.12	38.93
			4	28	12.87	16.03	24.03	58.59	117.99	55.17
		BOS PI S	1	39	19.30	36.02	67.95	207.03	240.13	117.80
			2	34	9.95	13.58	35.66	66.85	91.48	53.40
			3	32	7.47	9.06	20.78	45.87	66.89	39.15
			4	27	13.14	15.71	28.21	56.07	67.72	39.58
		BOS P-S	1	33	26.03	37.94	86.50	208.11	439.66	303.42
			2	35	7.79	10.61	30.87	96.70	135.64	85.32
			3	32	6.05	13.63	25.48	68.99	87.09	65.87
			4	21	8.68	13.90	37.08	65.97	73.78	55.25
	Window Dust Pb Conc.	BOS SPI	1	41	5840	8732	15262	28510	44187	22277
	($\mu g/g$)									

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Boston	Window Dust Pb Conc		2	41	4657	5533	12103	22756	24975	15250
	(μg/g)		3	38	3823	4957	21781	44533	53447	26723
			4	24	2983	4587	8780	16035	21950	14336
		BOS PI-S	1	41	3535	8299	22670	36022	47284	25565
			2	37	2824	5610	11527	30085	34197	15922
			3	37	2459	5879	18039	43803	52794	28975
			4	24	2023	3322	6870	10475	19267	8844
		BOS P-S	1	35	2421	4457	20057	60517	69988	30491
			2	37	2709	4542	17867	27941	50334	22537
			3	37	1441	1522	14601	46108	51872	26290
			4	19	2947	4457	12350	24050	24647	14060
	Window Dust Load	BOS SPI	1	41	70	133	295	630	796	450
	(mg/m^2)		2	41	122	249	440	706	913	592
			3	38	157	226	391	780	932	662
			4	24	228	385	919	1404	2579	1326
		BOS PI-S	1	41	106	159	304	522	757	624
			2	37	126	228	380	712	1174	583
			3	37	161	262	570	1095	1516	785
			4	24	92	155	500	766	993	556
		BOS P-S	1	35	74	142	239	444	629	494
			2	37	83	135	239	595	949	762
			3	37	91	239	504	990	1957	834
			4	19	169	185	797	976	1279	829

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Boston	Window Dust Pb Load	BOS SPI	1	41	1440	1953	7673	16401	20110	11143
	($\mu g/m^2$)		2	41	1050	1268	5228	9396	13373	7207
			3	38	1892	2944	6485	12797	19538	12792
			4	24	1252	1777	5402	20982	26748	14425
		BOS PI-S	1	41	1045	1819	8295	16998	24872	26774
			2	37	556	1657	5330	13312	17658	9326
			3	37	1388	3071	6566	15451	41927	17612
			4	24	701	1089	2553	6092	9175	5654
		BOS P	1	35	282	1294	4817	20608	28691	35798
			2	37	513	1303	5119	14086	16121	12164
			3	37	187	2906	6408	18833	36907	13412
			4	19	1569	1638	6018	28169	30796	12677
	Hand Pb Load	BOS SPI	1	54	9.4	11.00	13.00	17.00	17	14.97
	(μg/pair)		2	54	8.2	10.00	12.50	17.00	20	14.52
			3	53	8.8	13.00	17.00	21.00	23	18.06
			4	33	11.0	16.00	22.00	31.00	29	24.82
		BOS PI-S	1	51	10	11.00	13.00	15.00	20	13.97
			2	49	9	11.00	14.00	17.00	19	14.44
			3	46	12	9.30	15.50	20.00	29	18.10
			4	32	15	13.00	19.50	25.50	29	21.20
		BOS P-S	1	47	10	10.00	12.00	17.00	22	14.88
			2	46	9.1	9.80	12.00	18.00	20	16.18
			3	46	9.7	14.00	18.00	26.00	24	21.99
			4	26	9.7	16.00	20.00	26.00	37	22.64

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Boston	Blood Pb Conc	BOS SPI	1	54	8	10.00	13.00	16.00	18	13.19
	(μg/dL)		2	54	6	6.00	10.00	13.00	16	10.31
			3	54	6	7.00	10.00	14.00	16	11.70
			4	33	4	5.00	10.00	13.00	17	10.88
		BOS PI	1	51	8	9.00	12.00	15.00	17	12.37
			2	48	5	6.00	8.00	12.00	13	8.85
			3	49	8	9.00	11.00	14.00	16	11.49
			4	32	5	5.50	8.00	10.00	11	7.89
		BOS P	1	47	8	9.00	12.00	14.00	17	12.02
			2	46	6	8.00	9.00	12.00	14	9.83
			3	46	7	8.00	11.50	14.00	15	11.35
			4	26	5	6.00	10.00	13.00	13	9.96
Cincinnati	Soil Pb Conc	CIN SEI (P)	1	112	45	79	273	1190	1889	991
	(μg/g)		2	104	0	0	0	88	188	166
			3	104	17	18	28	64	138	132
			4	100	21	24	41	78	156	140
			5	100	23	25	38	112	187	163
			6	101	22	24	42	122	298	198
			7	103	23	25	37	117	194	167
		CIN I-SE (B)	1	26	25	42	89	107	122	122
			2	26	45	50	87	115	145	117
			3	26	53	58	93	131	144	153
			4	26	61	62	99	126	131	277
			5	26	40	49	64	93	95	67
			6	26	33	41	52	79	91	59
			7	26	35	37	55	77	92	68
		CIN I-SE (D)	1	-	-	-	-	-	-	-
			2	92	157	219	758	1561	2216	1141
			3	88	155	228	667	1400	1787	966

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Soil Pb Conc		4	86	155	307	768	1424	1928	1084
	(μg/g)		5	84	15	17	29	155	782	351
			6	82	19	22	41	156	823	334
			7	88	20	24	68	465	1002	508
		CIN I-SE (F)	1	46	56	104	608	1421	2034	1045
			2	48	63	125	760	1740	2142	1256
			3	49	76	108	294	1159	1795	817
			4	48	74	93	379	1109	1453	814
			5	48	16	21	37	605	1172	511
			6	48	17	19	70	1406	2557	929
			7	47	27	32	50	713	1231	835
		CIN NT (G)	1	118	0	8	69	221	357	176
			2	120	0	17	114	268	411	202
			3	120	31	46	124	308	520	573
			4	119	27	41	97	180	229	187
			5	120	34	43	99	216	347	192
			6	119	32	45	109	202	299	179
			7	121	26	38	111	197	313	169
		CIN NT (M)	1	44	52	100	349	1179	1728	809
			2	55	70	139	637	1376	1503	1013
			3	49	71	109	338	795	1120	654
			4	49	78	109	277	509	978	525
			5	48	90	110	349	673	976	613
			6	47	85	102	363	848	942	501
			7	48	63	132	416	860	1028	530
	Floor Dust Pb Conc.	CIN SEI (P)	1	30	197	247	366	610	805	566
	(μg/g)		2	30	224	239	362	585	680	463
			3	30	194	210	327	520	627	462
			4	25	222	286	478	608	913	617

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Floor Dust Pb Conc		5	24	0	0	0	0	0	0
	(µg/g)		6	-	-	-	-	-		-
			7	-	-	-	-	-		-
		CIN I-SE (B)	1	10	118	173	310	510	548	596
			2	9	252	351	403	499	1662	650
			3	8	304	324	480	637	638	469
			4	3	401	401	405	527	527	444
			5	1	0	0	0	0	0	0
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	23	236	333	414	533	618	441
			2	22	245	317	418	752	826	496
			3	23	192	250	414	574	623	448
			4	25	292	384	498	747	856	580
			5	21	0	0	0	0	0	0
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (F)	1	23	247	273	469	833	1233	897
			2	23	244	335	453	1291	1748	908
			3	22	267	290	392	640	757	526
			4	29	257	319	392	957	1191	1123
			5	22	0	0	0	0	0	0
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	31	93	125	187	332	475	269
			2	28	126	152	209	274	331	252
			3	29	125	145	184	259	302	216
			4	41	110	122	180	249	353	223
			5	35	0	0	0	0	0	0

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Floor Dust Pb Conc		7	-	-	-	-	-	-	-
	(µg/g)	CIN NT (M)	1	9	274	316	292	462	766	591
			2	9	213	341	562	699	724	594
			3	6	184	211	371	485	552	362
			4	14	219	255	434	660	772	781
			5	15	0	0	0	0	0	0
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Floor Dust Load	CIN SEI (P)	1	30	88	106	380	2248	4833	1714
	(mg/m^2)		2	30	36	53	136	397	1428	942
			3	30	83	93	135	307	358	285
			4	25	62	84	197	796	2123	784
			5	-	-	-	-	-	-	-
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (B)	1	10	45	57	127	1274	1433	1869
			2	9	11	22	31	53	103	45
			3	8	21	39	79	96	106	86
			4	3	85	85	137	266	266	163
			5	-	-	-	-	-	-	-
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	23	72	80	231	573	1269	791
			2	22	22	24	36	80	178	296
			3	23	47	86	119	263	331	205
			4	25	203	304	775	1745	3752	3289
			5	-	-	-	-	-	-	-
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Floor Dust Load	CIN I SE (F)	1	23	87	100	207	1140	3837	2839
	(mg/m^2)		2	23	21	31	52	103	122	136
			3	22	68	77	195	257	301	177
			4	29	154	293	420	623	986	704
			5	-	-	-	-	-	-	-
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	31	45	60	138	532	911	501
			2	28	49	79	138	209	275	327
			3	29	88	108	152	223	309	202
			4	41	67	102	196	345	646	499
			5	-	-	-	-	-	-	-
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (M)	1	9	86	105	207	319	332	258
			2	9	56	89	115	139	143	151
			3	6	14	98	258	333	438	233
			4	14	111	186	319	1161	1887	1102
			5	-	-	-	-	-	-	-
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Floor Dust Pb Load	CIN SEI (P)	1	30	20	32	168	1173	1645	867
	($\mu g/m^2$)		2	31	9	14	78	215	319	633
			3	30	25	27	54	101	129	180
			4	25	13	33	131	523	923	674
			5	24	6	34	77	264	580	323
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL.	Q1	Median (Q2)	Q3	84 PCTL.	Arithmetic Mean
Cincinnati	Floor Dust Pb Load	CIN I-SE (B)	1	10	12	12	40	417	657	4494
	($\mu g/m^2$)		2	9	4	5	9	26	171	47
			3	8	9	10	41	58	61	38
			4	3	34	34	55	140	140	77
			5	1	19	19	19	19	19	19
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	23	28	30	123	208	525	282
			2	22	6	8	19	36	53	37
			3	23	20	27	70	108	116	94
			4	25	116	145	298	1273	1579	1611
			5	21	25	48	149	544	750	434
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (F)	1	23	21	43	124	794	3022	5753
			2	23	9	12	28	64	180	102
			3	22	22	36	74	141	157	95
			4	29	60	112	176	546	945	662
			5	23	10	16	87	208	629	204
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	31	8	15	35	81	196	140
			2	28	12	14	29	64	78	78
			3	29	15	18	31	59	76	42
			4	41	10	15	22	86	233	118
			5	30	21	26	70	133	221	141
			6	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Floor Dust Pb Load		7	-	-	-	-	-	-	-
	($\mu g/m^2$)	CIN NT (M)	1	9	27	48	89	129	141	247
			2	9	20	25	59	81	81	143
			3	7	3	3	53	161	161	90
			4	14	80	81	128	513	1007	516
			5	14	47	52	141	438	598	284
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Window Dust Pb Conc.	CIN SEI (P)	1	30	507	729	1552	2672	3627	2775
	($\mu g/g$)		2	28	460	658	1309	1776	3144	3012
			3	28	540	674	929	1568	1919	1210
			4	25	796	987	1934	3373	6524	4165
			5	24	211	246	505	702	764	758
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (B)	1	10	726	2370	2803	3897	6575	4115
			2	8	519	603	806	1220	1331	2796
			3	8	472	578	1168	2749	3740	1695
			4	3	813	813	1077	3345	3345	1745
			5	1	1119	1119	1119	1119	1119	1119
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	23	886	1091	1835	2534	3214	2663
			2	18	700	1106	1969	2509	3100	1980
			3	23	633	1027	1318	2225	2229	1519
			4	25	1216	1377	1972	2761	2952	7180
			5	21	411	538	758	1305	1316	1313
			6							
			7							

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Window Dust Pb Conc	CIN I-SE (F)	1	21	511	1202	2015	2753	15853	7409
	(µg/g)		2	23	771	1165	2118	8285	14673	7488
			3	22	538	635	1442	2696	4723	2959
			4	29	1202	1355	2397	3323	7345	4309
			5	23	243	272	495	1047	1144	857
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	31	313	510	914	2222	3259	2151
			2	29	323	485	736	1446	2481	1347
			3	28	152	210	488	1216	1900	870
			4	41	414	543	1092	2096	2535	1495
			5	29	170	188	276	427	507	334
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (M)	1	9	381	920	1365	1903	4231	3637
			2	8	312	642	1467	4969	8314	5868
			3	7	292	292	643	1716	1716	1515
			4	15	1993	2058	3852	14612	17304	7416
			5	15	426	429	684	989	1126	1269
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Window Dust Load	CIN SEI (P)	1	30	52	137	729	3479	9217	10396
	($\mu g/m^2$)		2	28	96	112	443	1083	1563	3396
			3	28	80	110	254	507	675	531
			4	25	192	663	4524	21259	34180	20554
			5	24	553	613	966	1389	1699	1092
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Window Dust Load	CIN I-SE (B)	1	10	177	330	517	6659	8967	4436
	(mg/m^2)		2	8	61	62	222	830	1132	636
			3	8	68	100	179	630	702	355
			4	3	225	225	1514	34180	34180	11972
			5	1	164	164	164	164	164	164
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	23	316	544	1831	6146	14201	6147
			2	18	56	113	327	1230	9390	2719
			3	23	73	115	257	790	1200	798
			4	25	898	3574	7623	17658	34994	18088
			5	21	266	399	697	979	1189	1334
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (F)	1	21	113	178	1139	4381	13300	14711
			2	23	120	205	397	3748	5530	4223
			3	22	89	111	239	472	1203	1071
			4	29	935	4231	9632	17374	29250	15903
			5	23	248	329	649	977	1216	720
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	31	208	340	2621	5292	9615	4473
			2	29	89	316	2733	11895	20524	16777
			3	28	189	212	311	1040	1909	809
			4	41	1544	2767	8200	16956	31488	19333
			5	28	404	483	711	1198	1540	897
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Window Dust Lead	CIN NT (M)	1	9	349	409	1151	1412	2170	1660
	(mg/m^2)		2	8	66	80	474	4270	4298	1894
			3	7	200	200	405	681	681	418
			4	15	971	2164	3863	13530	19250	8526
			5	15	329	451	704	1139	1176	783
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Window Dust Pb Load	CIN SEI (P)	1	30	39	101	1083	9654	14707	35225
	($\mu g/m^2$)		2	31	48	81	488	1618	5112	67715
			3	28	84	149	243	380	1165	610
			4	25	287	386	15499	45741	86750	53635
			5	24	223	266	400	446	547	459
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (B)	1	10	344	492	1207	25185	25949	44601
			2	9	32	50	190	363	441	5375
			3	8	36	63	147	1658	2082	980
			4	3	753	753	1231	36808	36808	12931
			5	1	183	183	183	183	183	183
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	23	736	875	3056	10656	19575	51999
			2	22	37	60	196	3274	3627	5040
			3	23	68	131	353	1041	1818	1408
			4	25	1973	4145	14412	42127	110094	253299
			5	21	263	290	413	727	900	1120
			6	-	-	-				
			7	-	-	-				
		CIN I SE (F)	1	21	88	311	2619	13974	54618	371538

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Window Dust Pb Load		2	23	183	357	1137	12563	25653	14080
	($\mu g/m^2$)		3	22	129	174	355	1073	2044	2831
			4	29	1600	9845	29293	80592	101884	77791
			5	23	215	243	331	401	461	355
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	31	70	264	2080	8995	22246	12028
			2	29	122	217	1209	12089	47537	27483
			3	29	49	63	126	810	1103	1016
			4	41	824	2144	13777	29431	41924	26585
			5	29	133	141	217	250	309	241
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (M)	1	9	230	438	1731	1996	7362	3196
			2	9	34	64	360	6980	8711	6070
			3	7	58	58	361	694	694	678
			4	15	2763	4490	30480	84100	121949	62282
			5	15	244	276	381	721	752	508
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Entry Dust Pb Conc.	CIN SEI (P)	1	29	133	224	338	753	868	493
	($\mu g/g$)		2	31	383	432	666	957	1239	1118
			3	30	206	258	436	816	899	560
			4	24	119	264	495	873	1414	761
			5	24	25	44	213	1123	5397	1579
			6	22	202	209	384	588	770	443
			7	17	149	264	492	830	869	825
		CIN I-SE (B)	1	10	186	272	321	591	761	784

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Entry Dust Pb Conc		2	9	249	413	470	508	2388	855
	(µg/g)		3	8	337	366	445	564	573	512
			4	3	45	45	428	580	580	351
			5	1	56	56	56	56	56	56
			6	2	482	482	703	925	925	703
			7	1	279	279	279	279	279	279
		CIN I-SE (D)	1	22	272	361	528	758	1112	655
			2	22	356	384	636	1013	1148	695
			3	23	311	354	690	1042	1524	876
			4	25	475	590	831	1010	2012	1254
			5	21	73	95	184	661	1091	1365
			6	21	310	329	657	1208	1370	912
			7	18	292	395	708	899	982	735
		CIN I-SE (F)	1	22	241	299	563	872	1185	998
			2	22	367	465	630	1007	1463	1159
			3	22	231	270	410	755	973	1963
			4	29	224	303	390	814	1131	744
			5	23	19	21	67	118	1637	1241
			6	24	222	269	427	1173	1455	850
			7	18	217	384	478	779	856	554
		CIN NT (G)	1	30	87	119	243	363	447	306
			2	27	182	190	305	458	550	449
			3	29	193	222	309	362	478	332
			4	39	151	181	295	490	581	417
			5	29	18	28	56	280	453	606
			6	35	139	193	309	469	530	404
			7	31	205	223	270	442	469	338
		CIN NT (M)	1	9	334	355	473	621	1482	1918

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Entry Dust Pb Conc		2	9	392	407	577	811	1385	1053
	($\mu g/g$)		3	7	168	168	437	797	797	538
			4	15	328	455	883	3902	13096	3967
			5	15	69	101	355	952	3153	7878
			6	12	259	328	579	1423	2078	983
			7	9	285	482	642	1711	4891	1727
	Entry Dust Load	CIN SEI (P)	1	29	108	133	481	23077	98101	38881
	(mg/m^2)		2	31	35	48	114	2601	8344	6479
			3	30	112	145	230	837	1425	4920
			4	24	259	407	590	4060	26992	8088
			5	24	42	3118	12671	63462	92160	40218
			6	22	62	64	97	344	426	314
			7	17	105	143	301	1183	5020	2250
		CIN I-SE (B)	1	10	49	118	273	833	1543	197532
			2	9	29	36	49	79	323	1009
			3	8	56	80	284	357	371	258
			4	3	249	249	1156	42535	42535	14647
			5	1	48214	48214	48214	48214	48214	48214
			6	2	115	115	139	163	163	139
			7	1	260	260	260	260	260	260
		CIN I-SE (D)	1	22	56	88	375	863	1024	745
			2	22	31	39	59	125	144	159
			3	23	62	69	192	362	570	723
			4	25	377	419	2591	6266	14322	8989
			5	21	2493	3512	12796	27000	37500	21517
			6	21	83	93	179	339	622	1081

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Entry Dust Lead		7	18	214	261	534	1628	4160	1741
	(mg/m^2)	CIN I-SE (F)	1	22	81	101	346	1005	5537	3649
			2	22	38	63	116	203	285	169
			3	22	48	68	107	291	622	574
			4	29	130	244	913	4371	7605	9648
			5	23	359	11066	40299	128571	142105	62379
			6	24	59	80	182	926	2191	891
			7	18	109	199	632	3059	4856	3335
		CIN NT (G)	1	30	60	135	300	1312	2462	7776
			2	27	141	193	244	604	723	2002
			3	29	159	212	296	447	500	341
			4	39	165	236	435	3007	8824	34584
			5	29	3541	7521	34364	93103	150000	64155
			6	35	53	75	165	369	1789	3306
			7	31	190	367	952	1931	3388	26981
		CIN NT (M)	1	9	54	415	550	1776	3642	17224
			2	9	51	56	223	379	415	506
			3	7	65	65	222	299	299	233
			4	15	105	197	1341	7889	11616	4899
			5	15	424	660	4265	13745	27000	14109
			6	12	35	61	102	440	2989	662
			7	9	523	1020	1616	5417	14591	5298
	Entry Dust Pb Load	CIN SEI (P)	1	30	8	20	117	7009	31229	17609
	($\mu g/m^2$)		2	31	19	24	113	1385	14272	8693
			3	30	30	42	168	274	1171	2965
			4	25	38	140	252	2284	8496	11407

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Entry Dust Pb Load		5	24	326	963	2521	2720	2720	1944
	($\mu g/m^2$)		6	22	13	21	57	123	219	152
			7	17	27	34	151	907	2468	3646
		CIN I-SE (B)	1	10	9	40	82	493	524	892408
			2	9	12	12	18	97	164	2279
			3	8	41	52	92	201	213	117
			4	3	52	52	107	24681	24681	8280
			5	1	2720	2720	2720	2720	2720	2720
			6	2	79	79	93	107	107	93
			7	1	73	73	73	73	73	73
		CIN I-SE (D)	1	22	24	29	223	641	865	516
			2	22	19	20	40	55	75	155
			3	23	30	56	177	247	272	546
			4	25	349	359	2015	5037	8463	17160
			5	21	1922	2321	2720	2720	2720	2331
			6	21	43	61	118	319	353	710
			7	18	79	190	379	576	2770	2327
		CIN I-SE (F)	1	23	16	30	146	2375	4723	12898
			2	23	19	28	88	218	250	254
			3	22	16	19	44	204	1612	513
			4	29	77	102	308	2406	7275	10238
			5	23	588	1523	2720	2720	2720	2101
			6	24	29	45	126	538	907	668
			7	18	30	56	216	1864	4303	2500
		CIN NT (G)	1	31	13	16	72	305	482	1148
			2	28	30	42	83	219	514	513

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Entry Dust Pb Load		3	29	40	73	89	110	234	117
	($\mu g/m^2$)		4	40	33	49	155	959	3022	29691
			5	35	1523	1523	2720	2720	2720	2104
			6	35	13	16	46	211	499	1211
			7	31	41	118	242	641	1511	16089
		CIN NT (M)	1	9	12	250	387	1218	7052	25345
			2	9	36	36	74	177	1862	736
			3	7	44	44	81	177	177	100
			4	15	41	57	4477	12649	13532	26587
			5	15	292	453	1523	1889	2321	1414
			6	12	21	48	84	209	1310	439
			7	9	128	549	1544	2764	86636	21735
	Street Dust Pb Conc.	CIN SEI (P)	1	105	521	661	1286	2764	4127	2319
	($\mu g/g$)		2	85	515	757	1182	2024	2839	1900
			3	75	326	458	647	988	1526	1097
			4	66	453	684	994	2900	3603	1836
			5	89	601	749	1294	3171	3756	2386
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (B)	1	47	554	728	1407	1878	2275	1452
			2	47	334	643	1001	1656	1790	1172
			3	35	387	535	978	1331	1688	1927
			4	37	509	893	1298	1709	3191	1933
			5	42	758	955	1499	1966	2184	1836
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Street Dust Pb Conc	CIN I-SE (D)	1	20	635	680	1262	5384	5768	2782
	(µg/g)		2	22	566	696	1457	2056	2164	1488
			3	18	331	405	1011	1616	1956	1178
			4	19	439	461	1207	1766	1982	1491
			5	21	671	708	1024	4973	5245	2982
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (F)	1	33	1147	1283	1681	2553	4085	2371
			2	42	662	835	1274	2123	3631	2948
			3	34	525	715	1273	2406	5207	2239
			4	41	809	876	1520	3491	4832	2741
			5	42	1058	1197	2055	4606	6088	3793
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	15	75	118	212	343	348	238
			2	27	43	131	263	389	452	276
			3	14	117	132	229	325	352	261
			4	17	165	192	283	337	397	288
			5	14	84	86	162	357	367	234
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (M)	1	33	793	964	1217	1903	2138	1480
			2	35	520	512	584	785	1930	670
			3	35	473	512	584	785	865	670
			4	35	542	615	1157	1433	1801	1258
			5	35	562	655	884	1671	1908	1494

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Street Dust Pb Conc		6	-		-	-	-		-
	(µg/g)		7	-		-	-	-		-
	Sidewalk Dust Pb Conc.	CIN SEI (P)	1	84	788	1007	1809	4862	7565	3999
	(µg/g)		2	60	923	1240	2004	4622	8408	3748
			3	49	464	575	1478	2730	5307	3519
			4	48	773	930	1910	5779	7581	4150
			5	74	885	1078	2139	6310	8493	4534
			6	-	-	-	-	-	-	
			7	-	-	-	-	-		
		CIN I-SE (B)	1	61	765	1375	2376	4093	5777	3448
			2	44	745	1055	2330	4820	5983	3599
			3	36	790	1163	2691	4928	6171	3882
			4	37	808	1167	1646	5130	9525	3853
			5	45	887	1066	1899	3677	5232	3261
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	19	776	1037	1801	3087	4128	3899
			2	24	748	1228	2060	4577	5758	3577
			3	22	449	484	1294	5050	5325	2961
			4	20	669	794	2090	4738	5720	3413
			5	30	394	516	1696	3929	5890	3155
			6	-	-	-	-			-
			7	-	-	-	-	-	-	-
		CIN I-SE (F)	1	38	1471	1760	4456	9915	11342	6318
			2	45	1065	1420	3941	6215	7611	4727
			3	33	961	1284	3103	5587	9846	4839

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Sidewalk Dust Pb Conc		4	35	1177	1466	3365	9892	12393	6442
	(µg/g)		5	42	1307	1777	3125	5371	7334	4505
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	27	114	152	304	486	823	399
			2	41	101	158	297	541	631	390
			3	27	165	227	304	482	535	343
			4	25	146	178	315	369	490	314
			5	23	101	121	233	425	511	272
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (M)	1	37	495	938	1377	2539	3636	2310
			2	41	693	856	1410	4441	5683	2947
			3	36	533	686	1203	1999	2634	1886
			4	31	631	761	1101	4924	7706	3219
			5	34	477	564	1199	2714	2967	2448
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Hand Wipe Pb Load	CIN SEI (P)	1	51	4	5.00	6.00	11.00	14	12.14
	(µg/pair)		2	51	2	3.00	6.00	10.00	14	8.37
			3	35	2	3.00	5.00	8.00	10	5.94
			4	37	4	7.00	12.00	21.00	28	17.73
			5	30	4	7.00	12.50	19.00	24	16.03
		CIN I-SE (B)	1	24	4	5.00	17.50	33.00	43	24.17
			2	16	4	7.50	12.00	38.00	45	21.81
			3	11	4	4.00	7.00	10.00	11	6.45

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Hand Wipe Pb Load		4	5	2	2.00	2.00	5.00	6	3.40
	(μg/pair)		5	4	3	3.00	3.00	3.00	3	3.00
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	43	4	5.00	7.00	15.00	34	14.09
			2	43	3	3.00	7.00	14.00	24	11.19
			3	32	2	2.00	5.00	12.50	15	7.13
			4	41	5	8.00	12.00	29.00	35	18.93
			5	34	4	4.00	9.50	14.00	21	11.29
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (F)	1	30	2	4.00	6.50	13.00	19	11.57
			2	33	3	4.00	6.00	11.00	13	7.79
			3	30	2	3.00	5.50	9.00	11	7.67
			4	48	2	5.50	9.00	19.50	26	12.54
			5	34	4	6.00	8.00	16.00	29	13.97
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	46	1	1.00	3.00	5.00	6	4.74
			2	48	0	1.00	2.50	5.00	6	3.40
			3	34	0	1.00	3.00	4.00	5	2.88
			4	58	-1	0	3.50	7.00	10	4.52
			5	46	2	2.00	5.00	7.00	10	5.91
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (M)	1	10	1	2.00	7.00	18.00	20	10.60

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Hand Wipe Pb Load		2	11	1	6.00	10.00	15.00	18	10.09
	(µg/pair)		3	7	-2	-2	0	4.00	4	1.71
			4	24	2	4.50	8.50	19.00	25	14.96
			5	15	5	5.00	10.00	20.00	28	27.6
			6	-	-	-	-	-		
			7	-	-	-	-	-		
	Blood Pb Conc.	Cin SEI (P)	1	56	4.98	6.37	9.62	14.88	17.04	10.94
	(µg/dL)		3	52	3.46	4.69	7.00	10.74	13.42	8.26
			4	46	5.46	6.47	7.99	14.78	15.88	10.17
			6	37	5.05	6.04	7.89	10.54	14.89	9.31
			7	31	5.31	5.75	8.34	12.25	16.00	9.88
		CIN I SE (B)	1	24	7.33	8.46	13.12	21.39	26.16	15.19
			3	14	6.48	6.93	9.74	11.49	13.96	9.80
			4	8	7.14	7.61	9.40	11.70	13.00	9.53
			6	5	2.68	6.02	7.72	9.70	10.50	7.32
			7	2	7.97	7.97	8.43	8.90	8.90	8.43
		CIN I-SE (D)	1	44	7.51	8.65	12.59	17.96	20.14	13.85
			3	43	5.92	7.35	10.39	16.88	18.00	11.72
			4	47	4.90	5.96	10.40	14.98	17.95	12.00
			6	44	5.96	6.54	8.69	14.10	15.26	10.82
			7	43	6.80	7.03	8.98	17.19	21.89	13.09
		CIN I-SE (F)	1	35	6.45	7.60	9.45	14.28	16.10	11.41
			3	35	5.48	6.00	8.00	11.00	12.49	9.02
			4	49	3.63	4.67	7.59	13.38	20.05	10.12
			6	46	5.04	5.41	7.53	10.09	12.44	8.80
			7	36	5.65	6.96	8.82	12.31	19.04	11.70

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Blood Pb Conc	CIN NT (G)	1	46	5.05	5.96	8.48	10.72	12.32	9.02
	(μg/dL)		3	42	3.00	4.47	5.92	7.00	9.95	6.35
			4	62	2.80	3.57	5.32	7.27	10.08	6.11
			6	53	4.10	4.63	5.25	7.47	9.24	6.51
			7	48	4.58	5.31	6.19	8.22	9.49	7.21
		CIN NT (M)	1	12	2.50	6.69	11.84	16.91	19.15	11.63
			3	11	3.46	3.46	6.48	11.49	13.96	8.05
			4	24	7.17	8.03	11.91	15.33	19.35	12.44
			6	23	4.35	5.44	9.47	12.04	13.56	9.20
			7	15	7.17	7.39	8.59	16.40	17.55	11.30
Baltimore	Soil Pb Conc.	BAL SP	1	56	274	374	511	674	739	532
	(μg/g)		4	56	7	12	29	73	105	69
		BAL P-1	1	45	354	372	515	650	815	568
		BAL P-2	1	6	148	167	182	214	239	189
	Floor Dust Pb Conc.	BAL SP	1	64	542	884	1771	3495	5066	3226
	(μg/g)	BAL P-1	1	50	488	859	1939	3875	6966	6032
		BAL P-2	1	5	443	708	1130	2292	2448	1404
	Floor Dust Load	BAL SP	1	64	21.2	24.6	41.9	76.3	92.3	53.2
	(mg/m^2)	BAL P-1	1	50	12.3	16.9	33.1	48.1	60.7	39.0
		BAL P-2	1	5	26.0	60.4	120	122	123	90.2
	Floor Dust Pb Load	BAL SP	1	64	20.3	33.6	78.0	144	232	146
	($\mu g/m^2$)	BAL P-1	1	50	13.7	19.5	63.3	146	239	160
		BAL P-2	1	5	42.2	50.4	57.4	66.1	287	101

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Baltimore										
	Hand Wipe Load	BAL SP	1	78	4.62	5.67	8.63	13.73	18.26	11.45
	(µg/pair)		2	77	3.50	5.13	7.87	16.13	19.51	13.38
			3	104	2.20	4.00	6.80	10.47	15.70	8.84
			4	103	1.90	2.90	5.88	9.20	13.40	8.41
			5	99	4.00	6.00	11.00	17.00	24.00	13.01
			6	95	6.00	6.00	10.00	17.00	22.00	14.43
		BAL P-1	1	72	3.49	4.52	8.43	13.37	14.92	9.85
			2	72	4.38	5.29	9.05	15.02	21.58	15.08
			3	79	2.20	3.30	7.10	12.00	14.00	8.66
			4	79	2.20	2.70	4.30	6.90	9.10	6.11
			5	80	5.00	7.00	13.00	21.50	29.00	17.40
			6	79	5.00	6.00	10.00	15.00	19.00	12.78
		BAL P-2	1	7	5.08	5.08	6.24	14.09	14.09	9.39
			2	7	3.97	3.97	7.39	10.39	10.39	9.99
			3	8	2.80	3.05	6.65	8.04	9.00	6.40
			4	8	0	1.05	4.59	6.50	7.04	5.40
			5	7	9.00	9.00	13.00	19.00	19.00	15.86
			6	8	9.00	10.50	13.00	17.00	19.00	15.88
	Blood Pb Conc.	BAL SP	1	78	6.99	8.49	12.45	16.95	18.35	12.87
	(µg/dL)		2	77	6.55	8.00	10.75	13.85	16.35	11.87
			3	104	6.20	7.07	9.87	13.09	16.20	11.13
			4	103	5.63	6.40	8.65	11.55	14.04	9.73
			5	99	5.90	7.10	9.85	13.49	14.75	11.27

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Baltimore	Blood Pb Conc		6	95	6.25	7.45	10.35	12.70	14.70	**10.90**
	(μg/g)	BAL P-1	1	72	6.66	7.54	11.12	15.00	17.35	**12.37**
			2	72	6.30	7.20	10.34	13.65	15.45	**11.05**
			3	79	5.95	6.55	9.05	12.90	14.45	10.37
			4	79	4.74	5.54	7.40	10.85	12.85	9.02
			5	80	5.10	5.82	8.00	11.05	14.60	9.35
			6	79	4.90	5.70	8.10	11.90	15.00	9.73
		BAL P-2	1	7	6.85	6.85	10.85	13.50	13.50	10.42
			2	7	7.05	7.05	8.29	11.35	11.35	8.73
			3	8	4.95	5.07	7.77	8.72	9.45	7.22
			4	8	6.40	6.42	7.59	9.10	9.15	7.57
			5	7	5.49	5.49	8.15	10.05	10.05	8.11
			6	8	5.25	5.57	7.22	10.02	10.05	7.80

APPENDIX B:

THE P-VALUES FOR THE TABLES IN CHAPTER 5

TABLE B-1. P-VALUES FOR TABLE 5-1. PREABATEMENT CROSS-SECTIONAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY

	SEM EQUATION COEFFICIENTS		FLOOR DUST LEAD CONCENTRATION					
			Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	INTERCEPT	G_{gr}	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
SLOPE	Floor → Blood	B_{gr}		0.4447	0.3555		0.0027	
SLOPE	Soil → Blood	F_{gr}	0.5199		0.4423	0.5553		0.1150
SLOPE	Window → Blood	F_{gr}				0.2589	0.1231	
	INTERCEPT	C_{gr}	0.0037	0.0041	0.0035	0.0034	0.0031	0.0010
SLOPE	Soil → Floor	D_{gr}	0.5030	0.4149	0.5026	0.4957	0.4537	0.0107
SLOPE	Window → Floor	D_{gr}	0.0001	0.0001	0.0001	0.0001	0.0001	
			FLOOR DUST LEAD LOADING					
	INTERCEPT	G_{gr}	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
SLOPE	Floor → Blood	B_{gr}					0.4926	
SLOPE	Soil → Blood	F_{gr}	0.3774	0.3412	0.3459	0.4130	0.3658	0.1840
SLOPE	Window → Blood dust Pb conc	F_{gr}				0.2332		
SLOPE	Window → Blood dust Pb load	F_{gr}			0.4611			
	INTERCEPT	C_{gr}	0.2354	0.0338	0.0303	0.2435	0.0303	0.0044
SLOPE	Soil → Dust	D_{gr}	0.4051	0.6178	0.6436	0.3678	0.6295	0.0221
SLOPE	Window → Floor dust Pb conc	D_{gr}	0.0001			0.0001		
SLOPE	Window → Floor dust Pb load	D_{gr}		0.0008	0.0011		0.0011	

TABLE B-2. P-VALUES FOR TABLE 5-2. PREABATEMENT CROSS-SECTIONAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: BLOOD LEAD TRUNCATED (9-22 μg/dL)

	SEM EQUATION COEFFICIENTS		FLOOR DUST LEAD CONCENTRATION					
			Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	INTERCEPT	G_{gr}	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
SLOPE	Floor → Blood	B_{gr}		0.1849		0.9114	0.0178	
SLOPE	Soil → Blood	F_{gr}	0.1786	0.1457	0.1961	0.3622		0.1041
SLOPE	Window → Blood	F_{gr}			0.1697	0.8260	0.0806	
	INTERCEPT	C_{gr}	0.0217	0.0241	0.0235	0.0245	0.0125	0.0026
SLOPE	Soil → Floor	D_{gr}	0.3386	0.0360	0.3118	0.3143	0.3973	0.0270
SLOPE	Window → Floor	L_{gr}	0.0001	0.0001	0.0001	0.0001	0.0001	
			FLOOR DUST LEAD LOADING					
			Model 7	Model 8	Model 9	Model 10	Model 11	Model 12
	INTERCEPT	G_{gr}	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
SLOPE	Floor → Blood	B_{gr}			0.2954			
SLOPE	Soil → Blood	F_{gr}	0.1832	0.2025	0.1807	0.1929	0.1763	0.0699
SLOPE	Window → Blood dust Pb conc	F_{gr}				0.1271		
SLOPE	Window → Blood dust Pb load	F_{gr}					0.3898	
	INTERCEPT	C_{gr}	0.3562	0.0304	0.0288	0.3757	0.0282	0.0071
SLOPE	Soil → Dust	D_{gr}	0.3302	0.7126	0.7664	0.3395	0.7870	0.0451
SLOPE	Window → Floor dust Pb conc	L_{gr}	0.0001			0.0001		
SLOPE	Window → Floor dust Pb load	L_{gr}		0.0050	0.0043		0.0041	

TABLE B-3. P-VALUES FOR TABLE 5-3. PREABATEMENT CROSS-SECTIONAL STRUCTURAL EQUATION MODELS FOR CINCINNATI STUDY: DUST TYPE MODELS

SEM EQUATION COEFFICIENTS		With No Soil → Blood Slope			With Soil → Blood Slope			
		Floor	Entry	Window	Floor	Entry	Window	Soil
DUST LEAD CONCENTRATION ALL AGES								
INTERCEPT	G_g	0.0001	.0008	.0051	0.0031	.1603	.9967	0.0001
Slope: Dust → Blood	B	0.3647	.3081	.3947	0.9984	.0001	.9997	---
Slope: Soil → Blood	F	---	---	---	0.7063	.0706	.9995	0.3015
INTERCEPT	C_g	0.0001	.0001	.0001	0.0001	.0001	.0001	0.0001
Slope: Soil → Dust	D	0.0001	.0006	.0075	0.0001	.0009	.0075	0.0001
DUST LEAD LOADING ALL AGES								
INTERCEPT	G_g	.0001	.0001	.0001	.0001	.0001	.0001	.0001
Slope: Dust → Blood	B	.5829	.4486	.7328	.1529	.7449	.7918	---
Slope: Soil → Blood	F	---	---	---	.3231	.7617	.3478	.3421
INTERCEPT	C_g	.2788	.2644	.0657	.2964	.2536	.0759	.2943
Slope: Soil → Dust	D	.0051	.0409	.7441	.0049	.0506	.6443	.0049
DUST LEAD CONCENTRATION for AGE 42+ MONTHS								
INTERCEPT	G_g	.7219	.4987	.7807	.0001	.0002	.0002	.0001
Slope: Dust → Blood	B	.0046	.2169	.2881	0001	.0001	.0001	---
Slope: Soil → Blood	F	---	---	---	.0549	.0554	.0304	.0503
INTERCEPT	C_g	.0020	.0684	.0025	.0113	.0581	.0023	.0100
Slope: Soil → Dust	D	.0870	.0518	.0691	.0602	.2108	.0905	.0686
DUST LEAD LOAD for AGE 42+ MONTHS								
INTERCEPT	G_g	.0352	.0001	.0001	.0003	.1287	.0003	.0001
Slope: Dust → Blood	B	.2544	.0001	.0001	.0001	.0883	.0001	---
Slope: Soil → Blood	F	---	---	---	.0355	.7934	.0305	.0425
INTERCEPT	C_g	.3536	.2802	.3109	.4705	.3427	.3327	.3995
Slope: Soil → Dust	D	.0885	.7528	.8682	.0779	.6516	.9976	.0903

APPENDIX B:

THE P-VALUES FOR THE TABLES IN CHAPTER 5

TABLE B-1. P-VALUES FOR TABLE 5-1. PREABATEMENT CROSS-SECTIONAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY

	SEM EQUATION COEFFICIENTS		FLOOR DUST LEAD CONCENTRATION					
			Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	INTERCEPT	G_{gr}	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
SLOPE	Floor → Blood	B_{gr}		0.4447	0.3555		0.0027	
SLOPE	Soil → Blood	F_{gr}	0.5199		0.4423	0.5553		0.1150
SLOPE	Window → Blood	F_{gr}				0.2589	0.1231	
	INTERCEPT	C_{gr}	0.0037	0.0041	0.0035	0.0034	0.0031	0.0010
SLOPE	Soil → Floor	D_{gr}	0.5030	0.4149	0.5026	0.4957	0.4537	0.0107
SLOPE	Window → Floor	D_{gr}	0.0001	0.0001	0.0001	0.0001	0.0001	
			FLOOR DUST LEAD LOADING					
	INTERCEPT	G_{gr}	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
SLOPE	Floor → Blood	B_{gr}					0.4926	
SLOPE	Soil → Blood	F_{gr}	0.3774	0.3412	0.3459	0.4130	0.3658	0.1840
SLOPE	Window → Blood dust Pb conc	F_{gr}				0.2332		
SLOPE	Window → Blood dust Pb load	F_{gr}			0.4611			
	INTERCEPT	C_{gr}	0.2354	0.0338	0.0303	0.2435	0.0303	0.0044
SLOPE	Soil → Dust	D_{gr}	0.4051	0.6178	0.6436	0.3678	0.6295	0.0221
SLOPE	Window → Floor dust Pb conc	D_{gr}	0.0001			0.0001		
SLOPE	Window → Floor dust Pb load	D_{gr}		0.0008	0.0011		0.0011	

TABLE B-2. P-VALUES FOR TABLE 5-2. PREABATEMENT CROSS-SECTIONAL STRUCTURAL EQUATION MODELS FOR BOSTON STUDY: BLOOD LEAD TRUNCATED (9-22 μg/dL)

	SEM EQUATION COEFFICIENTS		FLOOR DUST LEAD CONCENTRATION					
			Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	INTERCEPT	G_{gr}	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
SLOPE	Floor → Blood	B_{gr}		0.1849		0.9114	0.0178	
SLOPE	Soil → Blood	F_{gr}	0.1786	0.1457	0.1961	0.3622		0.1041
SLOPE	Window → Blood	F_{gr}			0.1697	0.8260	0.0806	
	INTERCEPT	C_{gr}	0.0217	0.0241	0.0235	0.0245	0.0125	0.0026
SLOPE	Soil → Floor	D_{gr}	0.3386	0.0360	0.3118	0.3143	0.3973	0.0270
SLOPE	Window → Floor	L_{gr}	0.0001	0.0001	0.0001	0.0001	0.0001	
			FLOOR DUST LEAD LOADING					
			Model 7	Model 8	Model 9	Model 10	Model 11	Model 12
	INTERCEPT	G_{gr}	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
SLOPE	Floor → Blood	B_{gr}			0.2954			
SLOPE	Soil → Blood	F_{gr}	0.1832	0.2025	0.1807	0.1929	0.1763	0.0699
SLOPE	Window → Blood dust Pb conc	F_{gr}				0.1271		
SLOPE	Window → Blood dust Pb load	F_{gr}					0.3898	
	INTERCEPT	C_{gr}	0.3562	0.0304	0.0288	0.3757	0.0282	0.0071
SLOPE	Soil → Dust	D_{gr}	0.3302	0.7126	0.7664	0.3395	0.7870	0.0451
SLOPE	Window → Floor dust Pb conc	L_{gr}	0.0001			0.0001		
SLOPE	Window → Floor dust Pb load	L_{gr}		0.0050	0.0043		0.0041	

TABLE B-3. P-VALUES FOR TABLE 5-3. PREABATEMENT CROSS-SECTIONAL STRUCTURAL EQUATION MODELS FOR CINCINNATI STUDY: DUST TYPE MODELS

SEM EQUATION COEFFICIENTS		With No Soil → Blood Slope			With Soil → Blood Slope			
		Floor	Entry	Window	Floor	Entry	Window	Soil
DUST LEAD CONCENTRATION ALL AGES								
INTERCEPT	G_g	0.0001	.0008	.0051	0.0031	.1603	.9967	0.0001
Slope: Dust → Blood	B	0.3647	.3081	.3947	0.9984	.0001	.9997	---
Slope: Soil → Blood	F	---	---	---	0.7063	.0706	.9995	0.3015
INTERCEPT	C_g	0.0001	.0001	.0001	0.0001	.0001	.0001	0.0001
Slope: Soil → Dust	D	0.0001	.0006	.0075	0.0001	.0009	.0075	0.0001
DUST LEAD LOADING ALL AGES								
INTERCEPT	G_g	.0001	.0001	.0001	.0001	.0001	.0001	.0001
Slope: Dust → Blood	B	.5829	.4486	.7328	.1529	.7449	.7918	---
Slope: Soil → Blood	F	---	---	---	.3231	.7617	.3478	.3421
INTERCEPT	C_g	.2788	.2644	.0657	.2964	.2536	.0759	.2943
Slope: Soil → Dust	D	.0051	.0409	.7441	.0049	.0506	.6443	.0049
DUST LEAD CONCENTRATION for AGE 42+ MONTHS								
INTERCEPT	G_g	.7219	.4987	.7807	.0001	.0002	.0002	.0001
Slope: Dust → Blood	B	.0046	.2169	.2881	.0001	.0001	.0001	---
Slope: Soil → Blood	F	---	---	---	.0549	.0554	.0304	.0503
INTERCEPT	C_g	.0020	.0684	.0025	.0113	.0581	.0023	.0100
Slope: Soil → Dust	D	.0870	.0518	.0691	.0602	.2108	.0905	.0686
DUST LEAD LOAD for AGE 42+ MONTHS								
INTERCEPT	G_g	.0352	.0001	.0001	.0003	.1287	.0003	.0001
Slope: Dust → Blood	B	.2544	.0001	.0001	.0001	.0883	.0001	---
Slope: Soil → Blood	F	---	---	---	.0355	.7934	.0305	.0425
INTERCEPT	C_g	.3536	.2802	.3109	.4705	.3427	.3327	.3995
Slope: Soil → Dust	D	.0885	.7528	.8682	.0779	.6516	.9976	.0903

TABLE B-4. P-VALUES FOR TABLE 5-4. PREABATEMENT CROSS-SECTIONAL STRUCTURAL EQUATION MODELS FOR CINCINNATI STUDY: FLOOR DUST

SEM EQUATION COEFFICIENTS		FLOOR DUST LEAD CONCENTRATION							
		Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
INTERCEPT G_{gr}		0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0031
SLOPE	Floor → Blood		0.0405	0.0486	0.9455	0.4629		0.3647	0.9984
	Soil → Blood	0.4202		0.8660	0.7143		0.3015		0.7063
	Window → Blood	0.0347			0.7622	0.9645			
INTERCEPT G_{gr}		0.0037	0.0036	0.0037	0.0037	0.0037	0.0001	0.0001	0.0001
SLOPE	Soil → Floor	0.0009	0.0006	0.0009	0.0009	0.0009	0.0001	0.0001	0.0001
	Window → Floor	0.0002	0.0002	0.0002	0.0002	0.0002			

		FLOOR DUST LEAD LOADING				
		Model 9	Model 10	Model 11	Model 12	Model 13
INTERCEPT G_{gr}		0.0001		0.0001	0.0001	0.0001
SLOPE	Floor → Blood		0.4052		0.5829	0.1529
	Soil → Blood	0.4339	0.4177	0.3421		0.3231
	Window → Blood	0.2831	0.3439			
INTERCEPT G_{gr}		0.5756	0.5611	0.2943	0.2788	0.2964
SLOPE	Soil → Dust	0.0026	0.0025	0.0049	0.0051	0.0049
	Window → Floor	0.0001	0.0001			

TABLE B-5. P-VALUES FOR TABLE 5-5. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BOSTON STUDY: EFFECT OF AGE REDUCTION IN BLOOD LEAD (E_r) BETWEEN ROUNDS 1 AND 3

Study Group		Age Group			
		All Ages (N=150)	0-17 Months (N=19)	18-41 Months (N=100)	42+ Months (N=31)
Abate	Control	Response (E_r)			
BOS SPI	BOS P-S	0.0042	0.7312	0.0020	0.4271
BOS PI-S	BOS P-S	0.6157	0.3844	0.1280	0.6163
BOS SPI	BOS PI-S	0.0159	0.2019	0.0769	0.2320
		Log Response (E_r)			
BOS SPI	BOS P-S	0.0064	0.7543	0.0084	0.2465
BOS PI-S	BOS P-S	0.6414	0.4093	0.2917	0.9614
BOS SPI	BOS PI-S	0.0212	0.2307	0.0809	0.3186

TABLE B-6. P-VALUES FOR TABLE 5-6. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BOSTON STUDY: EFFECT OF AGE REDUCTION IN BLOOD LEAD (E_r) BETWEEN ROUNDS 3 AND 4

Study Group		Age Group			
		All Ages (N=147)	0-17 Months (N=18)	18-41 Months (N=98)	42+ Months (N=31)
Abate	Control	Response (E_r)			
BOS P-S	BOS SPI	0.1222	0.7834	0.5365	0.1172
BOS PI-S	BOS SPI	0.0006	0.4907	0.0138	0.1007
BOS PI-S	BOS P-S	0.0788	0.6295	0.0761	0.9819
		Log Response (E_r)			
BOS P-S	BOS SPI	0.3772	0.6609	0.8137	0.2065
BOS PI-S	BOS SPI	0.0217	0.6127	0.0736	0.3125
BOS PI-S	BOS P-S	0.1931	0.8366	0.1280	0.7795

TABLE B-7. P-VALUES FOR TABLE 5-7. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BOSTON STUDY: EFFECT OF RACE OR SEX

Study Group			Group				
		Rounds 1-3:	All (N = 150)	Black (N = 75)	Nonblack (N = 32)	Male (N = 80)	Female (N = 70)
		Rounds 3-4:	(N = 147)	(N = 74)	(N = 32)	(N = 78)	(N = 69)
Abate	Control		Reduction in Blood Lead (E_T) Between Rounds 1 and 3				
BOS SPI	BOS P-S		0.0042	0.0502	0.2477	0.0162	0.1306
BOS PI-S	BOS P-S		0.6157	0.4116	0.9031	0.9105	0.5665
BOS SPI	BOS PI-S		0.0159	0.2670	0.1136	0.0205	0.3213
			Reduction in Blood Lead (E_T) Between Rounds 3 and 4				
BOS P-S	BOS SPI		0.1222	0.0136	0.5241	0.2881	0.2198
BOS PI-S	BOS SPI		0.0006	0.0044	0.3577	0.0498	0.0039
BOS P-S	BOS P-S		0.0788	0.5798	0.1990	0.4091	0.0929
			Reduction in Log Blood Lead (E_T) Between Rounds 1 and 3				
BOS SPI	BOS P-S		0.0064	0.0644	0.6097	0.0099	0.1796
BOS PI-S	BOS P-S		0.6414	0.2843	0.4840	0.9756	0.5870
BOS SPI	BOS PI-S		0.0212	0.4432	0.1178	0.0099	0.3987
			Reduction in Log Blood Lead (E_T) Between Rounds 3 and 4				
BOS P-S	BOS SPI		0.3772	0.0136	0.9262	0.2881	0.2198
BOS PI-S	BOS SPI		0.0217	0.0044	0.6607	0.0498	0.0039
BOS P-S	BOS P-S		0.1931	0.5798	0.8046	0.4091	0.0929

TABLE B-8. P-VALUES FOR TABLE 5-8. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BOSTON STUDY: EFFECT OF TRUNCATION REDUCTION IN BLOOD LEAD (E_r) BETWEEN ROUNDS 1 AND 3

Study Group			Truncation Category			
		Age 18-41:	7-24 μg/dL (N=100)	10-19 μg/dL (N=67)	7-19 μg/dL (N=92)	10-24 μg/dL (N=75)
		Age 42-52:	(N=31)	(N=16)	(N=29)	(N=18)
Abate	Control		Change in Blood Lead for Age Group 18-41 Months			
BOS SPI	BOS P-S		0.0020	0.0129	0.0039	0.0070
BOS PI-S	BOS P-S		0.1280	0.3616	0.2665	0.1847
BOS SPI	BOS PI-S		0.0769	0.1021	0.0590	0.1250
			Change in Blood Lead for Age Group 42-52 Months			
BOS SPI	BOS P-S		0.4271	0.7966	0.2969	1.0000
BOS PI-S	BOS P-S		0.6163	0.4695	0.8885	0.2901
BOS SPI	BOS PI-S		0.2320	0.3448	0.2733	0.2901
			Change in Log Blood Lead for Age Group 18-41 Months			
BOS SPI	BOS P-S		0.0084	0.0336	0.0115	0.0270
BOS PI-S	BOS P-S		0.2917	0.5450	0.3828	0.4433
BOS SPI	BOS PI-S		0.0809	0.1189	0.0810	0.1143
			Change in Log Blood Lead for Age Group 42-52 Months			
BOS SPI	BOS P-S		0.2465	0.7846	0.1917	0.9389
BOS PI	BOS P-S		0.9614	0.3759	0.8064	0.2618
BOS SPI	BOS PI-S		0.3186	0.2644	0.3302	0.2367

TABLE B-9. P-VALUES FOR TABLE 5-9. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BOSTON STUDY: EFFECT OF TRUNCATION REDUCTION IN BLOOD LEAD (E_r) BETWEEN ROUNDS 3 AND 4

Study Group		Truncation Category			
		7-24 μg/dL (N=31)	10-19 μg/dL (N=16)	7-19 μg/dL (N=29)	10-24 μg/dL (N=18)
Abate	Control	Change in Blood Lead for Age Group 18-41 months			
BOS P-S	BOS SPI	0.5365	0.2526	0.7232	0.1696
BOS PI-S	BOS SPI	0.0138	0.0330	0.0788	0.0040
BOS PI-S	BOS P-S	0.0765	0.3025	0.1618	0.1368
		Change in Log Blood Lead for Age Group 18-41 Months			
BOS P-S	BOS SPI	0.8137	0.2430	0.9443	0.1739
BOS PI-S	BOS SPI	0.0736	0.0377	0.1479	0.0093
BOS P-S	BOS P-S		0.3431	0.1689	0.2295

TABLE B-10. P-VALUES FOR TABLE 5-10. REPEATED MEASURES ANALYSIS OF VARIANCE FOR CINCINNATI STUDY: EFFECT OF AGE IN BETWEEN ROUNDS 1 AND 4

Study Group		Age Group			
		All Ages (N=223)	9-17 Months (N=69)	18-41 Months (N=80)	42+ Months (N=70)
Abate	Control	Reduction in Blood Lead (E_T) Between Rounds 1 and 4			
CIN NT (G)	CIN NT (M)	0.0729	0.0014	0.0722	0.1783
CIN SEI (P)	CIN NT(G)	0.0365	0.0199	0.5882	0.1992
CIN SEI (P)	CIN NT (M)	0.6023	0.0534	0.1038	0.0173
CIN SEI (P)	CIN I-SE (D)	0.0492	0.5731	0.9563	0.0028
CIN SEI (P)	CIN I-SE (F)	0.3640	0.7196	0.8852	0.0818
CIN I-SE (D)	CIN NT (G)				
CIN I-SE (D)	CIN NT (M)				
CIN I-SE (F)	CIN NT (G)				
CIN I-SE (F)	CIN NT (M)				
		Reduction in Log Blood Lead (E_T) Between Rounds 1 and 4			
CIN NT (G)	CIN NT (M)	0.0032	0.0001	0.0669	0.5310
CIN SEI (P)	CIN NT(G)	0.0016	0.0046	0.1986	0.0384
CIN SEI (P)	CIN NT (M)	0.2959	0.0098	0.1618	0.0219
CIN SEI (P)	CIN I-SE (D)	0.0595	0.6488	0.9666	0.0078
CIN SEI (P)	CIN I-SE (F)	0.2139	0.5151	0.7619	0.0676
CIN I-SE (D)	CIN NT (G)				
CIN I-SE (D)	CIN NT (M)				
CIN I-SE (F)	CIN NT (G)				
CIN I-SE (F)	CIN NT (M)				

TABLE B-11. P-VALUES FOR TABLE 5-11. REPEATED MEASURES ANALYSIS OF VARIANCE FOR CINCINNATI STUDY: EFFECT OF AGE IN BETWEEN ROUNDS 4 AND 7

Study Group		Age Group			
		All Ages (N = 223)	9-17 Months (N = 69)	18-41 Months (N = 80)	42 + Months (N = 70)
Abate	Control	Reduction in Blood Lead (E_r) Between Rounds 4 and 7			
CIN NT (G)	CIN NT (M)	0.1440	0.3869	0.7044	
CIN SEI (P)	CIN NT(G)	0.4298	0.6781	0.8856	
CIN SEI (P)	CIN NT (M)	0.3201	0.5018	0.7442	
CIN SEI (P)	CIN I-SE (D)	0.9920	0.7354	0.6530	
CIN SEI (P)	CIN I-SE (F)	0.5164	0.5826	0.8982	
		Reduction in Log Blood Lead (E_r) Between Rounds 4 and 7			
CIN NT (G)	CIN NT (M)	0.0622		0.6735	0.1227
CIN SEI (P)	CIN NT(G)	0.1257		0.4815	0.1866
CIN SEI (P)	CIN NT (M)	0.3429		0.8921	0.6276
CIN SEI (P)	CIN I-SE (D)	0.7484		0.6539	0.3885
CIN SEI (P)	CIN I-SE (F)	0.6793		0.7254	0.5682

TABLE B-12. P-VALUES FOR TABLE 5-12. REPEATED MEASURES ANALYSIS OF VARIANCE FOR CINCINNATI STUDY: EFFECT OF TRUNCATION BETWEEN ROUNDS 1 AND 4

Study Group Abate Versus Control		Truncation Category			
		All	7-24 μg/dL	10-19 μg/dL	10-24 μg/dL
Abate	Control	Reduction in Blood Lead (E_r) Age 9-17 Months			
		(N=69)	(N=33)	(N=15)	
CIN NT (G)	CIN NT (M)	0.0014			
CIN SEI (P)	CIN NT(G)	0.0199	0.4899	0.8627	
CIN SEI (P)	CIN NT (M)	0.0534			
CIN SEI (P)	CIN I-SE (D)	0.5731	0.6991	0.8599	
CIN SEI (P)	CIN I-SE (F)	0.7196	0.4300	0.6409	
		Reduction in Blood Lead (E_r) Age 18-41 Months			
		(N=80)	(N=67)	(N=38)	(N=43)
CIN NT (G)	CIN NT (M)	0.0722	0.1018	0.0527	0.0521
CIN SEI (P)	CIN NT(G)	0.5882	0.6432	0.2596	0.3909
CIN SEI (P)	CIN NT (M)	0.1038	0.1366	0.1417	0.1048
CIN SEI (P)	CIN I-SE (D)	0.9563	0.5682	0.4924	0.3518
CIN SEI (P)	CIN I-SE (F)	0.8852	0.6966	0.7628	0.8940
		Reduction in Blood Lead (E_r) Age 42+ Months			
		(N=70)	(N=47)	(N=31)	(N=36)
CIN NT (G)	CIN NT (M)	0.1783	0.4788	0.5605	0.6069
CIN SEI (P)	CIN NT(G)	0.1992	0.2337	0.5561	0.6233
CIN SEI (P)	CIN NT (M)	0.0173	0.0594	0.2629	0.3350
CIN SEI (P)	CIN I-SE (D)	0.0028	0.0107	0.2353	0.1276
CIN SEI (P)	CIN I-SE (F)	0.0818	0.3625	0.9944	0.9834

TABLE B-13. P-VALUES FOR TABLE 5-13. REPEATED MEASURES ANALYSIS OF VARIANCE FOR CINCINNATI STUDY: EFFECT OF TRUNCATION BETWEEN ROUNDS 1 AND 4

Study Group Abate Versus Control		Truncation Category		
		All	7-24 μg/L	10-19 μg/L
Abate	Control	Reduction in Log Blood Lead (E_T) Age 9-17 Months		
		(N=69)	(N=33)	(N=15)
CIN NT (G)	CIN NT (M)	0.0001		
CIN SEI (P)	CIN NT(G)	0.0046	0.6858	0.9889
CIN SEI (P)	CIN NT (M)	0.0098		
CIN SEI (P)	CIN I-SE (D)	0.6488	0.9496	0.7852
CIN SEI (P)	CIN I-SE (F)	0.5151	0.2463	0.7416
		Reduction in Log Blood Lead (E_T) Age 18-41 Months		
		(N=80)	(N=67)	(N=38)
CIN NT (G)	CIN NT (M)	0.0669	0.1757	
CIN SEI (P)	CIN NT(G)	0.1986	0.4559	
CIN SEI (P)	CIN NT (M)	0.1618	0.2726	
CIN SEI (P)	CIN I-SE (D)	0.9666	0.7355	
CIN SEI (P)	CIN I-SE (F)	0.7619	0.6738	
		Reduction in Log Blood Lead (E_T) Age 42+ Months		
		(N=70)	(N=47)	(N=31)
CIN NT (G)	CIN NT (M)	0.5310		
CIN SEI (P)	CIN NT(G)	0.0384	0.1589	
CIN SEI (P)	CIN NT (M)	0.0219	0.0941	
CIN SEI (P)	CIN I-SE (D)	0.0078	0.0147	
CIN SEI (P)	CIN I-SE (F)	0.0676	0.3864	

TABLE B-14. P-VALUES FOR TABLE 5-14. REPEATED MEASURES ANALYSIS OF VARIANCE FOR CINCINNATI STUDY: EFFECT OF TRUNCATION BETWEEN ROUNDS 4 AND 7

Study Group Abate Versus Control		Truncation Category			
		All	7-24 μg/dL	10-19 μg/dL	10-24 μg/L
Abate	Control	Reduction in Blood Lead (E_T) Age 9-17 Months			
		(N=69)	(N=33)	(N=15)	
CIN NT (G)	CIN NT (M)	0.3869			
CIN SEI (P)	CIN NT(G)	0.6781	0.9241	0.8588	
CIN SEI (P)	CIN NT (M)	0.5018			
CIN SEI (P)	CIN I-SE (D)	0.7354	0.4648	0.7252	
CIN SEI (P)	CIN I-SE (F)	0.5826	0.3079	0.9711	
		Reduction in Blood Lead (E_T) Age 18-41 Months			
		(N=80)	(N=67)	(N=38)	(N=43)
CIN NT (G)	CIN NT (M)	0.7044	0.8282	0.6802	0.9919
CIN SEI (P)	CIN NT(G)	0.8856	0.8166	0.8214	0.7274
CIN SEI (P)	CIN NT (M)	0.7442	0.7475	0.7602	0.8558
CIN SEI (P)	CIN I-SE (D)	0.6530	0.5430	0.6156	0.5004
CIN SEI (P)	CIN I-SE (F)	0.8982	0.8460		0.8702
		Reduction in Blood Lead (E_T) Age 42+ Months			
		(N=70)	(N=47)	(N=31)	(N=36)
CIN NT (G)	CIN NT (M)		0.2750	0.3188	0.2820
CIN SEI (P)	CIN NT(G)		0.2785	0.3822	0.3515
CIN SEI (P)	CIN NT (M)		0.9935	0.9229	0.9220
CIN SEI (P)	CIN I-SE (D)		0.5976	0.6980	0.6659
CIN SEI (P)	CIN I-SE (F)		0.8317	0.8346	0.8277

TABLE B-15. P-VALUES FOR TABLE 5-15. REPEATED MEASURES ANALYSIS OF VARIANCE FOR CINCINNATI STUDY: EFFECT OF TRUNCATION BETWEEN ROUNDS 4 AND 7

Study Group Abate Versus Control		Truncation Category			
		All	7-24 (μg/dL)	10-19 (μg/dL)	10-24 μg L
Abate	Control	Reduction in Log Blood Lead (E_T) Age 9-17 Months			
		(N=69)	(N=33)	(N=15)	
CIN NT (G)	CIN NT (M)				
CIN SEI (P)	CIN NT(G)		0.5801		
CIN SEI (P)	CIN NT (M)				
CIN SEI (P)	CIN I-SE (D)		0.7779		
CIN SEI (P)	CIN I-SE (F)		0.8283		
		Reduction in Log Blood Lead (E_T) Age 18-41 Months			
		(N=80)	(N=67)	(N=38)	(N=43)
CIN NT (G)	CIN NT (M)	0.6735	0.8043		
CIN SEI (P)	CIN NT(G)	0.4815	0.8345		
CIN SEI (P)	CIN NT (M)	0.8921	0.8715		
CIN SEI (P)	CIN I-SE (D)	0.6539	0.5018		
CIN SEI (P)	CIN I-SE (F)	0.7254	0.6618		
		Reduction in Log Blood Lead (E_T) Age 42+ Months			
		(N=70)	(N=47)	(N=31)	(N=36)
CIN NT (G)	CIN NT (M)	0.1227	0.5405		
CIN SEI (P)	CIN NT(G)	0.1866	0.4497		
CIN SEI (P)	CIN NT (M)	0.6276	0.8869		
CIN SEI (P)	CIN I-SE (D)	0.3885	0.5114		
CIN SEI (P)	CIN I-SE (F)	0.5682	0.7		

TABLE B-16. P-VALUES FOR TABLE 5-16. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BALTIMORE STUDY: EFFECT OF AGE

Study Group		Age Group			
		All Ages (N=463)	< 18 Months (N=16)	18-41 Months (N=88)	42+ Months (N=161)
Abate	Control	Reduction in Blood Lead (E_1) Between Rounds 3 and 4			
BAL SP	BAL P1	0.8893	0.2790	0.8040	0.9097
BAL SP	BAL P2	0.1625		0.3593	0.1168
		Reduction in Blood Lead (E_T) Between Rounds 3 and 6			
BAL SP	BAL P1	0.3676	0.9313	0.6495	0.7862
BAL SP	BAL P2	0.6456		0.1654	0.6910
		Reduction in Log Blood Lead (E_1) Between Rounds 3 and 4			
BAL SP	BAL P1	0.8563			0.9761
BAL SP	BAL P2	0.9882			0.3854
		Reduction in Log Blood Lead (E_T) Between Rounds 3 and 6			
BAL SP	BAL P1	0.8420			
BAL SP	BAL P2	0.9683			

TABLE B-17. P-VALUES FOR TABLE 5-17. REPEATED MEASURES ANALYSIS OF VARIANCE FOR BALTIMORE STUDY: EFFECT OF TRUNCATION BETWEEN ROUNDS 3 AND 4

		TRUNCATION CATEGORY				
		ALL <18: (N=16) 18-42: (N=88) >42: (N=161)	10-19 <18: (N=2) 18-42: (N=32) >42: (N=47)	10-24 <18: (N=4) 18-42: (N=42) >42: (N=53)	7-19 <18: (N=15) 18-42: (N=54) >42: (N=110)	7-24 <18: (N=7) 18-42: (N=64) >42: (N=120)
STUDY GROUP						
ABATE	CONTROL	REDUCTION IN BLOOD LEAD (E_T) FOR AGE <18 MONTHS				
BAL SP	BAL P1					
BAL SP	BAL P2					
		REDUCTION IN BLOOD LEAD (E_T) FOR AGE 18-41 MONTHS				
BAL SP	BAL P1	0.8040	0.3455		0.1255	0.6136
BAL SP	BAL P2	0.3593				
		REDUCTION IN BLOOD LEAD (E_T) FOR AGE 42+ MONTHS				
BAL SP	BAL P1	0.9097	0.7233	0.8511	0.7934	0.8338
BAL SP	BAL P2	0.1168			0.2096	0.3194
		REDUCTION IN LOG BLOOD LEAD (E_T) FOR AGE <18 MONTHS				
BAL SP	BAL P1					
BAL SP	BAL P2					
		REDUCTION IN LOG BLOOD LEAD (E_T) FOR AGE 18-41 MONTHS				
BAL SP	BAL P1					
BAL SP	BAL P2					
		REDUCTION IN LOG BLOOD LEAD (E_T) FOR AGE 42+ MONTHS				
BAL SP	BAL P1	0.9761	0.5998	0.6122	0.4523	0.774(
BAL SP	BAL P2	0.3854			0.4184	0.4700

TABLE B-18. P-VALUES FOR TABLE 5-18. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD CONCENTRATION REDUCTION IN LOG BLOOD LEAD (E_r) BETWEEN ROUNDS 1 AND 3

Study Group Abate Versus Control		Age Group			
		All Ages (N=142)	9-17 Months (N=17)	18-41 Months (N=97)	42+ Months (N=28)
BOS SPI	BOS P-S	0.8014	0.8276	0.3125	0.4279
BOS PI-S	BOS P-S	0.1341	0.3036	0.1323	0.5034
BOS SPI	BOS PI-S	0.2106	0.4492	0.5690	0.8213
Covariate: Log Dust Pb Concentration					
BOS SPI	BOS P-S	0.9442	0.8363	0.4275	0.4013
BOS PI-S	BOS P-S	0.1334	0.2589	0.1440	0.4962
BOS SPI	BOS PI-S	0.1672	0.4131	0.4895	0.8216

TABLE B-19. P-VALUES FOR TABLE 5-19. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD AND SOIL LEAD CONCENTRATION REDUCTION IN LOG BLOOD LEAD (E_r) BETWEEN ROUNDS 1 AND 3

Study Group Abate Versus Control		Age Group			
		All Ages (N=142)	9-17 Months (N=17)	18-41 Months (N=97)	42+ Months (N=28)
BOS SPI	BOS P-S			0.1845	0.7040
BOS PI-S	BOS P-S			0.5148	0.5661
BOS SPI	BOS PI-S			0.5601	0.6962
Covariate: Log Dust Lead Concentration					
BOS SPI	BOS P-S			0.4880	0.4556
BOS PI-S	BOS P-S			0.1557	0.6810
BOS SPI	BOS PI-S			0.4426	0.9828
Covariate: Log Soil Lead Concentration					
BOS SPI	BOS P-S			0.5469	0.8314
BOS PI-S	BOS P-S			0.6789	0.6742
BOS SPI	BOS PI-S			0.2891	0.5028

TABLE B-20. P-VALUES FOR TABLE 5-20. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR: BOSTON STUDY EFFECT OF AGE AND LOG DUST LEAD CONCENTRATION REDUCTION IN LOG BLOOD LEAD (E_T) BETWEEN ROUNDS 3 AND 4

Study Group Abate Versus Control		Age Group			
		All Ages (N = 142)	9-17 Months (N = 17)	18-41 Months (N = 97)	42 + Months (N = 28)
BOS SPI	BOS P-S	0.6993	0.4752	0.1995	0.5128
BOS PI-S	BOS P-S	0.0367		0.0291	0.8716
BOS SPI	BOS PI-S	0.0275		0.0026	0.5167
Covariate: Log Dust Lead Concentration					
BOS SPI	BOS P-S	0.7205	0.4608		0.4667
BOS PI-S	BOS P-S	0.0245			0.9146
BOS SPI	BOS PI-S	0.0206			0.5090

TABLE B-21. P-VALUES FOR TABLE 5-21. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD LOADING REDUCTION IN BLOOD LEAD (E_T) BETWEEN ROUNDS 3 AND 4

Study Group Abate Versus Control		Age Group			
		All Ages (N = 128)	9-17 Months (N = 15)	18-41 Months (N = 89)	42 + Months (N = 24)
BOS SPI	BOS P-S	0.8045		0.5540	0.5013
BOS PI-S	BOS P-S	0.2436		0.0558	0.2273
BOS SPI	BOS PI-S	0.1774		0.1251	0.1596
Covariate: Log Dust Lead Loading					
BOS SPI	BOS P-S	0.9244		0.5885	0.7092
BOS PI-S	BOS P-S	0.0788		0.0167	0.1821
BOS SPI	BOS PI-S	0.0700		0.0370	0.1568

TABLE B-22. P-VALUES FOR TABLE 5-22. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD CONCENTRATION ON BLACKS REDUCTION IN LOG BLOOD LEAD (E_r) BETWEEN ROUNDS 1 AND 3

Study Group Abate Versus Control		Age Group			
		All Ages (N=71)	9-17 Months (N=11)	18-41 Months (N=44)	42+ Months (N=16)
BOS SPI	BOS P-S	0.5834		0.2446	0.5699
BOS PI-S	BOS P-S	0.0185		0.0070	0.4295
BOS SPI	BOS PI-S	0.0694		0.0663	0.6882
Covariate: Log Dust Lead Concentration					
BOS SPI	BOS P-S	0.6832		0.3190	0.5484
BOS PI-S	BOS P-S	0.0188		0.0078	0.4414
BOS SPI	BOS PI-S	0.0583		0.0583	0.7043

TABLE B-23. P-VALUES FOR TABLE 5-23. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD LOAD ON BLACKS REDUCTION IN LOG BLOOD LEAD (E_r) BETWEEN ROUNDS 1 AND 3

Study Group Abate Versus Control		Age Group			
		All Ages (N=71)	9-17 Months (N=11)	18-41 Months (N=44)	42+ Months (N=16)
BOS SPI	BOS P-S	0.7408		0.2904	0.6548
BOS PI-S	BOS P-S	0.2493		0.0482	0.7336
BOS SPI	BOS PI-S	0.4199		0.2902	0.6941
Covariate: Log Dust Lead Load					
BOS SPI	BOS P-S	0.8594		0.6841	0.6595
BOS PI-S	BOS P-S	0.3266		0.1386	0.6151
BOS SPI	BOS PI-S	0.3000		0.2902	0.7171

TABLE B-24. P-VALUES FOR TABLE 5-24. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR BOSTON STUDY: EFFECT OF AGE AND LOG DUST LEAD CONCENTRATION, SOIL LEAD CONCENTRATION ON BLACKS REDUCTION IN BLOOD LEAD (E_r) BETWEEN ROUNDS 1 AND 3

Study Group Abate Versus Control		Age Group			
		All Ages (N=71)	9-17 Months (N=11)	18-41 Months (N=44)	42+ Months (N=16)
BOS SPI	BOS P-S	0.6991		0.3049	
BOS PI-S	BOS P-S	0.3351		0.5147	
BOS SPI	BOS PI-S	0.5369		0.8734	
Covariate: Log Dust Lead Concentration					
BOS SPI	BOS P-S	0.6574		0.2806	
BOS PI-S	BOS P-S	0.0220		0.0068	
BOS SPI	BOS PI-S	0.0603		0.0451	
Covariate: Log Soil Lead Concentration					
BOS SPI	BOS P-S	0.8834		0.9902	
BOS PI-S	BOS P-S	0.4605		0.1370	
BOS SPI	BOS PI-S	0.5550		0.1267	

TABLE B-25. P-VALUES FOR TABLE 5-25. REPEATED MEASURES ANALYSIS OF COVARIANCE EFFECT OF AGE AND LOG DUST LEAD LOADING ON BLACKS IN BOSTON STUDY REDUCTION IN LOG BLOOD LEAD (E_r) BETWEEN ROUNDS 3 AND 4

Study Group Abate Versus Control		Age Group			
		All Ages (N=128)	9-17 Months (N=15)	18-41 Months (N=89)	42+ Months (N=24)
BOS SPI	BOS P-S	0.6347		0.4728	0.4505
BOS PI-S	BOS P-S	0.3690		0.0479	0.1304
BOS SPI	BOS PI-S	0.2207		0.1276	0.1004
Covariate: Log Dust Lead Loading					
BOS SPI	BOS P-S	0.8309		0.4090	0.6792
BOS PI-S	BOS P-S	0.1594		0.0152	0.1042
BOS SPI	BOS PI-S	0.1431		0.0707	0.0983

TABLE B-26. P-VALUES FOR TABLE 5-26. REPEATED MEASURES ANALYSIS OF COVARIANC EEFFECT OF AGE AND LOG DUST LEAD CONCENTRATION ON BLACKS IN BOSTON STUDY REDUCTION IN LOG BLOOD LEAD (E_r) BETWEEN ROUNDS 3 AND 4

Study Group Abate Versus Control		Age Group			
		All Ages (N=64)	9-17 Months (N=8)	18-41 Months (N=40)	42+ Months (N=16)
BOS SPI	BOS P-S	0.8096		0.9335	0.9820
BOS PI-S	BOS P-S	0.1268		0.0483	0.4692
BOS SPI	BOS PI-S	0.1316		0.1710	0.5004
Covariate: Log Dust Lead Concentration					
BOS SPI	BOS P-S	0.8977		0.9555	0.9438
BOS PI-S	BOS P-S	0.0859		0.0349	0.4962
BOS SPI	BOS PI-S	0.1251		0.1695	0.4878

TABLE B-27. P-VALUES FOR TABLE 5-27. REPEATED MEASURES ANALYSIS OF COVARIANCE IN CINCINNATI STUDY REDUCTION IN BLOOD LEAD (E_r) BETWEEN ROUNDS 1 AND 4

Study Group		Log Floor Dust Concentration	Log Entry Dust Concentration	Log Window Dust Concentration	Log Floor Dust Pb Loading	Log Entry Dust Pb Loading	Log Window Dust Pb Loading
Abate Versus Control		Intercept Effect					
CIN NT(G)	CIN NT(M)	.8510	.3061	.0949	.1403	.0494	.8739
CIN SEI (P)	CIN NT(G)	.7127	.6939	.9510	.6924	.0815	.1331
CIN SEI (P)	CIN NT(M)	.7360	.3876	.1110	.1861	.3416	.4395
CIN SEI (P)	CIN I-SE(D)	.4674	.8180	.3970	.5635	.0610	.0144
CIN SEI (P)	CIN I-SE(F)	.7149	.5748	.9169	.2227	.2581	.6430
		Covariate Effect					
CIN NT(G)	CIN NT(M)	.9754	.4978	.0496	.2925	.2720	.5571
CIN SEI (P)	CIN NT(G)	.5155	.9633	.7537	.8128	.3314	.3686
CIN SEI (P)	CIN NT(M)	.8118	.4789	.0803	.2221	.6066	.3659
CIN SEI (P)	CIN I-SE(D)	.3838	.9945	.4317	.3684	.1133	.0251
CIN SEI (P)	CIN I-SE(F)	.6102	.6906	.9691	.2797	.3975	.6734

TABLE B-28. P-VALUES FOR TABLE 5-30. LONGITUDINAL STRUCTURAL EQUATION MODELS REGRESSION COEFFICIENTS IN BOSTON STUDY FREE BLOOD LEAD PERSISTENCE FACTOR

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17
RESPONSE VARIABLE: BLOOD LEAD ROUND 3						
Intercept	ALL GROUPS	0.4767	0.7504	0.8488	0.8468	
	BOS SPI					0.8530
	BOS PI-S					0.5376
	BOS P-S					0.4464
Soil Pb Round 3	ALL GROUPS	0.3463		0.9943	0.9943	0.9746
	BOS SPI		0.9693			
	BOS PI-S		0.4404			
	BOS P-S		0.7376			
Dust Pb Conc. Round 3	ALL GROUPS	0.7575	0.8584			0.0055
	BOS SPI			0.9944	0.9942	
	BOS PI-S			0.7467	0.7466	
	BOS P-S			0.8365	0.8341	
Blood Lead Round 1		0.0710	0.1794	0.4960	0.4942	0.3964
RESPONSE VARIABLE: BLOOD LEAD ROUND 1						
Intercept		0.0001	0.0001	0.0001	0.0001	0.0001
Soil Pb Round 1		0.1934	0.1831	0.6006	0.6042	0.1672
Dust Pb Round 1		0.0191	0.0400	0.8170	0.8209	0.0183
RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1						
Intercept		0.0001	0.0001	0.0001	0.0022	0.0002
Soil Pb Round 1		0.1110	0.1921	0.6307	0.6234	0.9375
Window Dust Pb Round 1		0.0001	0.0001	0.0001	0.0001	0.0001
RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 3						
Intercept	ALL GROUPS	0.0001	0.0001	0.0001		0.0001
	BOS SPI				0.0001	
	BOS PI-S				0.6734	
	BOS P-S				0.8706	
Soil Pb Conc. Round 3	ALL GROUPS	0.0001	0.0001	0.0001		0.0003
	BOS SPI				0.8352	
	BOS PI-S				0.4355	
	BOS P-S				0.2045	
Window Dust Pb Conc. Round 3		0.0077	0.0135	0.0076		0.0697

TABLE B-29. P-VALUES FOR TABLE 5-32. LONGITUDINAL STRUCTURAL EQUATION MODELS REGRESSION COEFFICIENTS IN BOSTON STUDY USING FIXED BLOOD LEAD PERSISTENCE FACTOR

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30
RESPONSE VARIABLE: BLOOD LEAD ROUND 3							
Intercept	ALL GROUPS	0.0269	0.0318	0.1524	0.1021		
	BOS SPI					0.8045	0.9768
	BOS PI-S					0.0327	0.0119
	BOS P-S					0.0269	0.0200
Soil Pb Round 3	ALL GROUPS	0.1503		0.6593	0.3903	0.5743	0.3162
	BOS SPI		0.6401				
	BOS PI-S		0.1294				
	BOS P-S		0.0757				
Floor Dust Pb Conc. Roung 3	ALL GROUPS	0.0568	0.1917			0.0468	0.0437
	BOS SPI			0.1311	0.2348		
	BOS PI-S			0.0507	0.0221		
	BOS P-S			0.0291	0.0312		
Blood Pb Round 1							
RESPONSE VARIABLE: BLOOD LEAD ROUND 1							
Intercept		0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
Soil Pb Round 1		0.3330	0.3255	0.4958	0.8490	0.4735	0.7502
Dust Pb Round 1		0.7917	0.7907	0.5487	0.5383	0.6413	0.6100
RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1							
Intercept		0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
Soil Pb Round 1		0.1372	0.1323	0.2802	0.1824	0.3020	0.1927
Window Dust Pb Round 1		0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 3							
Intercept	ALL GROUPS	0.0001	0.0001	0.0001		0.0001	
	BOS SPI				0.0001		0.0001
	BOS PI-S				0.8698		0.5948
	BOS P-S				0.3021		0.5005
Soil Pb Conc. Round 3	ALL GROUPS	0.0001	0.0001	0.0001		0.0001	
	BOS SPI				0.3886		0.3506
	BOS PI-S				0.2298		0.3651
	BOS P-S				0.3883		0.2386
Window Dust Pb Conc. Round 3		0.0079	0.0082	0.0062	0.0157	0.0107	0.0166

TABLE B-30. P-VALUES FOR TABLE 5-34. LONGITUDINAL STRUCTURAL EQUATION MODELS REGRESSION COEFFICIENTS IN BOSTON STUDY USING FIXED BLOOD LEAD PERSISTENCE FACTOR FOR MALES

Predictor Variable		REGRESSION COEFFICIENT					
		MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30
		RESPONSE VARIABLE: BLOOD LEAD ROUND 3					
Intercept	ALL GROUPS	0.0002	0.0086	0.0513	0.0088		
	BOS SPI					0.1630	0.0717
	BOS PI-S					0.0120	0.0031
	BOS P-S					0.1089	0.0285
Soil Pb Round 3	ALL GROUPS	0.0001		0.3791	0.9339	0.3859	0.9301
	BOS SPI		0.6309				
	BOS PI-S		0.0001				
	BOS P-S		0.0307				
Floor Dust Pb Conc. Round 3	ALL GROUPS	0.4309	0.4235			0.1455	0.2619
	BOS SPI			0.1533	0.6422		
	BOS PI-S			0.3364	0.3385		
	BOS P-S			0.1506	0.3398		
Blood Pb Round 1							
		RESPONSE VARIABLE: BLOOD LEAD ROUND 1					
Intercept		0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
Soil Pb Round 1		0.0034	0.0042	0.0127	0.4435	0.0061	0.3653
Dust Pb Round 1		0.8835	0.6506	0.7607	0.3940	0.6529	0.3225
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1					
Intercept		0.0001	0.0001	0.0001	0.0609	0.0001	0.0479
Soil Pb Round 1		0.2664	0.2448	0.3570	0.412	0.3282	0.0440
Window Dust Pb Round 1		0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 3					
Intercept	ALL GROUPS	0.0001	0.0001	0.0001		0.0001	
	BOS SPI				0.0001		0.0001
	BOS PI-S				0.362		0.0573
	BOS P-S				0.7345		0.7002
Soil Pb Conc. Round 3	ALL GROUPS	0.0001	0.0001	0.0001		0.0001	
	BOS SPI				0.2240		0.2593
	BOS PI-S				0.8994		0.7696
	BOS P-S				0.0243		0.0207
Window Dust Pb Conc. Round 3		0.3830	0.5289	0.6532	0.0267	0.3282	0.0415

TABLE B-31. P-VALUES FOR TABLE 5-36. LONGITUDINAL STRUCTURAL EQUATION MODELS REGRESSION COEFFICIENTS IN BOSTON STUDY USING FIXED BLOOD LEAD PERSISTENCE FACTOR FOR FEMALES

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30
		RESPONSE VARIABLE: BLOOD LEAD ROUND 3					
Intercept	ALL GROUPS	0.1339	0.0463	0.0713	0.8430		
	BOS SPI					0.1088	0.7959
	BOS PI-S					0.0283	0.4893
	BOS P-S					0.0016	0.4179
Soil Pb Round 3	ALL GROUPS	0.1015		0.8050	0.8166	0.4828	0.7927
	BOS SPI		0.5711				
	BOS PI-S		0.3810				
	BOS P-S		0.1018				
Floor Dust Pb Conc. Round 3	ALL GROUPS	0.0422	0.2234			0.9175	0.0029
	BOS SPI			0.4793	0.0337		
	BOS PI-S	. .		0.0549	0.0003		
	BOS P-S			0.0427	0.0031		
Blood Pb Round 1							
		RESPONSE VARIABLE: BLOOD LEAD ROUND 1					
Intercept		0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
Soil Pb Round 1		0.9857	0.9177	0.8133	0.3281	0.8552	0.3401
Dust Pb Round 1		0.1127	0.1609	0.3534	0.0609	0.2571	0.0443
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1					
Intercept		0.0001	0.0001	0.0002	0.0003	0.0003	0.0003
Soil Pb Round 1		0.0586	0.1121	0.1854	0.1529	0.2059	0.1702
Window Dust Pb Round 1		0.0034	0.0013	0.0001	0.0006	0.0001	0.0009
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 3					
Intercept	ALL GROUPS	0.0001	0.0001	0.0001		0.0001	
	BOS SPI				0.2191		0.2695
	BOS PI-S				0.6655		0.6518
	BOS P-S				0.6745		0.6479
Soil Pb Conc. Round 3	ALL GROUPS	0.0086	0.0156	0.0398		0.0192	
	BOS SPI				0.1871		0.1515
	BOS PI-S				0.9655		0.9565
	BOS P-S				0.9279		0.9338
Window Dust Pb Conc. Round 3		0.7834	0.7313	0.6324	0.2873	0.5848	0.3209

TABLE B-32. P-VALUES FOR TABLE 5-38. LONGITUDINAL STRUCTURAL EQUATION MODELS REGRESSION COEFFICIENTS IN BOSTON STUDY USING FIXED BLOOD LEAD PERSISTENCE FACTOR FOR AGES 18-41 MONTHS

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30	UNITS
		RESPONSE VARIABLE: BLOOD LEAD ROUND 3						
Intercept	ALL GROUPS	0.0074	0.0290	0.0935	0.6496			
	BOS SPI					0.6781	0.7693	
	BOS PI-S					0.0079	0.0209	
	BOS P-S					0.0093	0.0166	
Soil Pb Round 3	ALL GROUPS	0.2395		0.2787	0.0460	0.2337	0.0797	
	BOS SPI		0.4125					
	BOS PI-S		0.2681					
	BOS P-S		0.5112					
Floor Dust Pb Conc. Round 3	ALL GROUPS	0.2386	0.2375			0.0710	0.0442	
	BOS SPI			0.2091	0.1066			
	BOS PI-S			0.0451	0.0033			
	BOS P-S			0.0452	0.0087			
Blood Pb Round 1								
		RESPONSE VARIABLE: BLOOD LEAD ROUND 1						
Intercept		0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	
Soil Pb Round 1		0.9807	0.8312	0.7984	0.6676	0.7772	0.8262	
Dust Pb Round 1		0.7040	0.9488	0.7331	0.4729	0.9239	0.7176	
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1						
Intercept		0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	
Soil Pb Round 1		0.2118	0.1550	0.4371	0.8325	0.4744	0.7428	
Window Dust Pb Round 1		0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 3						
Intercept	ALL GROUPS	0.0001	0.0001	0.0001		0.0001		
	BOS SPI				0.0001		0.0001	
	BOS PI-S				0.6513		0.7169	
	BOS P-S				0.8737		0.9172	

TABLE B-32. P-VALUES FOR TABLE 5-38 (cont'd). LONGITUDINAL STRUCTURAL EQUATION MODELS FOR: REGRESSION COEFFICIENTS IN BOSTON STUDY USING FIXED BLOOD LEAD PERSISTENCE FACTOR FOR AGES 18-41 MONTHS

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 2	MODEL 10	MODEL 11	MODEL 17	MODEL 30	UNITS
Soil Pb Conc. Round 3	ALL GROUPS	0.0001	0.0001	0.0001		0.0001		
	BOS SPI				0.0065		0.0136	
	BOS PI-S				0.8239		0.8635	
	BOS P-S				0.6024		0.6551	
Window Dust Pb Conc. Round 3		0.0036	0.0033	0.0106	0.0011	0.0127	0.0030	

TABLE B-33. P-VALUES FOR TABLE 5-40. LONGITUDINAL STRUCTURAL EQUATION MODELS REGRESSION COEFFICIENTS IN CINCINNATI STUDY USING FIXED BLOOD LEAD PERSISTENCE FACTOR

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 5	MODEL 6	MODEL J5	MODEL J6	UNITS
		RESPONSE VARIABLE: BLOOD LEAD ROUND 4					
Intercept	ALL GROUPS	0.0050	0.0827		0.4888		
	CIN I-SE(D)			0.8904		0.2241	
	CIN I-SE(F)			0.1178		0.4976	
	CIN NT(G)			0.0328		0.3122	
	CIN NT(M)			0.0469		0.0304	
	CIN SEI(P)			0.9332		0.0919	
Floor Dust Pb Round 4		0.0038	0.0020	0.0001	0.0012	0.0011	
Soil Pb Round 4		0.9681	0.6385		0.5356		
Blood Pb Round 1 (Fixed)							
		RESPONSE VARIABLE: BLOOD LEAD ROUND 1					
Intercept	ALL GROUPS	0.0001	0.0001	0.0001			
	CIN I-SE(D)				0.0001	0.0001	
	CIN I-SE(F)				0.0001	0.0001	
	CIN NT(G)				0.0001	0.0001	
	CIN NT(M)				0.0001	0.0001	
	CIN SEI(P)				0.0001	0.0001	
Floor Dust Pb Round 1		0.481	0.0801	0.0230	0.0004	0.0023	
Soil Pb Round 1		0.8027	0.0460	0.0677			
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1					
Intercept	ALL GROUPS	0.0001	0.0001	0.0001			
	CIN I-SE(D)				0.0001	0.0002	
	CIN I-SE(F)				0.0659	0.1975	
	CIN NT(G)				0.0119	0.0223	
	CIN NT(M)				0.0001	0.0001	
	CIN SEI(P)				0.0011	0.0029	
Window Dust Pb Round 1		0.0002	0.0006	0.0003	0.0001	0.0001	
Soil Pb Round 1		0.0025	0.0089	0.0040			

TABLE B-33. P-VALUES FOR TABLE 5-40 (cont'd). LONGITUDINAL STRUCTURAL EQUATION MODELS REGRESSION COEFFICIENTS IN CINCINNATI STUDY USING FIXED BLOOD LEAD PERSISTENCE FACTOR

Predictor Variable		REGRESSION COEFFICIENT MODEL 1	MODEL 5	MODEL 6	MODEL J5	MODEL J6	UNITS
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 1					
Intercept	ALL GROUPS						
	CIN I-SE(D)						
	CIN I-SE(F)						
	CIN NT(G)						
	CIN NT(M)						
	CIN SEI(P)						
Window Dust Pb Round 1							
Soil Pb Round 1							
		RESPONSE VARIABLE: FLOOR DUST LEAD CONCENTRATION ROUND 4					
Intercept	ALL GROUPS	0.0001					
	CIN I-SE(D)		0.0001	0.0001	0.0001	0.0001	
	CIN I-SE(F)		0.0001	0.0002	0.0001	0.0009	
	CIN NT(G)		0.0002	0.0001	0.0001	0.0001	
	CIN NT(M)		0.0818	0.4535	0.4119	0.4972	
	CIN SEI(P)		0.0001	0.0001	0.0001	0.0001	
Window Dust Pb Round 1		0.0001	0.0001	0.0001	0.0001	0.0001	
Soil Pb Round 1		0.0107					

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